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Table of Contents.

INTUBATION, ENTEROSTOMY, AND LAPAROTOMY: THEIR PLACE IN THE TREATMENT OF INTESTINAL OBSTRUCTION.

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CONSIDERABLE difference of opinion exists as to the appropriate place of intubation suction decompression, enterostomy and laparotomy in the treatment of the obstructed bowel. In general, the older school of surgeons leans to early operation though accepting a short period of intubation suction as a valuable pre-operative measure.

At the other extreme are those who believe that intubation should be used in certain cases for prolonged periods with the object of obtaining resolution of the obstruction. Each of these schools tends to regard intubation suction and enterostomy as alternative and competitive methods of decompressing distended bowel. The correct viewpoint is that intubation, enterostomy and laparotomy all have their place, and are to be used alone, successively or in conjunction according to definite indications.

INTUBATION DECOMPRESSION.

The requirements for intubation decompression are an appropriate tube and a system for applying suction to such tube.

Two types of tube are available: (i) The short single-lumen tube for gastric or duodenal intubation includes the Rehfuss, Ryle, Levin and Wangensteen tubes. The Wangensteen tube, being graduated, and having a rounded, heavy, lead-impregnated catheter tip facilitating introduction and a relatively large lumen, is most useful when gastric content containing food or large flocculi requires aspiration. (ii) The long intestinal tube for intubation of the small bowel includes the Miller-Abbott, the Harris (Harris, 1945; Herrara, Millet and Lawrence, 1947; Harris and Gordon, 1948), the Cantor (Cantor, Kennedy and Reynolds, 1947), and the Honor-Smathers (Honor and Smathers, 1947) tubes (Figure I). When it appears that aspiration may be required for a prolonged period, an intestinal tube is used in preference to one of the first group, even though it may be felt that short-tube suction alone may prove adequate. The discomfort is greater only during the process of the tube's passage into the stomach. This is more than compensated for by the many advantages resulting from intestinal intubation.

A disadvantage of the Miller-Abbott tube is the small size of the suction lumen relative to the tube's external diameter, owing to the necessary presence of the second lumen for filling the balloon. This is an appreciable disadvantage, as blockage of the lumen is apt to occur in the early stages of decompression, and during oral feeding after satisfactory decompression has been attained. Nevertheless, provided adequate attention is given to the apparatus, such disability is not material.

Harris and Cantor eliminate the second lumen by attaching to a single-lumen tube a rubber bag containing a small amount of mercury. The weight and fluidity of the mercury facilitate the migration of the tube through the pylorus.

In the Harris tube, the rubber tube passes through the centre of the bag and projects beyond it. In the Cantor tube the bag hangs from the tip of the tube.

In both, the contents of the bag cannot be varied during intubation, the onward passage of the bag depending on its weight and not on its bulk. The absence of bulk is a disadvantage.

The Honor-Smathers tube combines the good features of the above-mentioned tubes. The bag is terminal, hanging from the tip of the tubing, and its contents may be varied during intubation owing to the use of double-lumen tubing. The bag-filling lumen is smaller than that of the

Miller-Abbott tube and the suction lumen is correspondingly larger. The writer modifies a Miller-Abbott tube to resemble the Honor-Smathers type.

The Advantages of Long-Tube Suction.

Those who use, as a pre-operative measure, gastric or duodenal suction, find that such great improvement frequently results that they question whether intestinal suction is of sufficient additional value to warrant its routine use.

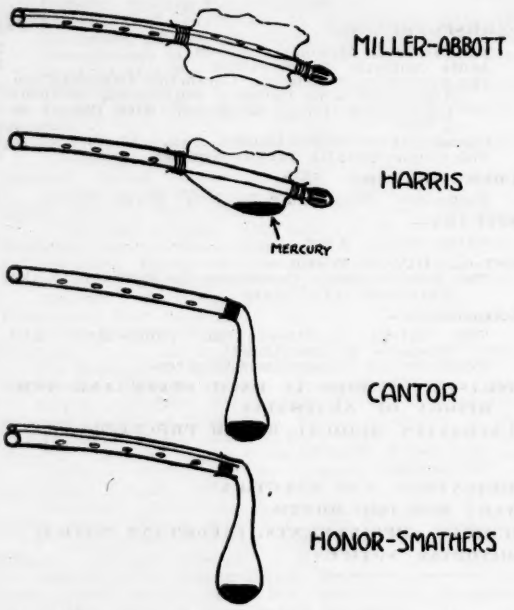


FIGURE I.

Long intestinal tubes. Only in the Miller-Abbott and Honor-Smathers tubes may the contents of the bag be altered during intubation. The Honor-Smathers tube has the additional advantage of a soft terminal bag hanging from the tip of the tube.

Some very material advantages of intestinal suction are as follows:

1. Decompression is more rapidly and more efficiently attained. Case reports quoted later give instances in which gastric suction, after a few days, has been replaced by intestinal intubation and in these the acceleration of decompression is obvious. With gastric suction, unless free regurgitation of intestinal content into the stomach occurs, gastric mucus tends to thicken the gastric contents and so block the tube. It is noticeable, once the tube tip has entered the duodenum, that the fluid is always thinner and runs more readily. Apart from this, as the tube migrates, it successively opens up kinked bowel loops.

2. Oral feedings, while the tube tip is in the stomach or duodenum, are valueless. However, once the tube is past the duodeno-jejunal flexure, even though decompression is incomplete, a non-residue diet may be given simultaneously with suction. Digestion and absorption may be deficient early, but rapidly become adequate as the tube tip descends, so allowing discontinuance of intravenous feeding. (Cases II and III.)

3. Decompression by a long intestinal tube is usually so efficient that when operation is performed on a non-strangulated obstruction, the patient's general condition is commonly excellent; in most cases the bowel is flaccid and collapsed and may be readily packed where required and the lesion is readily accessible, with healthy circulation and amenable to the most radical of treatment. It is common to find that not only may one operative stage (that of surgical decompression) be eliminated, but also that intraabdominal resection and anastomosis may be performed safely, in cases in which, after other methods of pre-operative preparation, an exteriorizing resection would have been advisable. (Cases II, VIII and XI.) The intestinal tube may be threaded down to any suture line, and thus afford during the first few post-operative days direct protection of this line while at the same time allowing of oral feeding.

4. In extensive adhesive obstruction, palpation of the tube through the bowel wall frequently provides a useful indication as to which is the more proximal part of the bowel. (Cases I, VII and VIII.) In a number of these cases, after separation of multiple adhesions and the production of raw surfaces that could not be adequately peritonealized, the tube has been threaded well down the bowel in the hope that, if adhesions did recur, they would do so in the bowel, not subjected initially at any rate to acute kinking.

5. Instillation of thin barium solution via the tube with simultaneous fluoroscopy may (but not frequently) give useful information as to the nature of the obstruction, or its partial character as indicated by the passage of barium beyond the obstruction (Case III). (Figures VIII and IX.)

The Suction Apparatus.

It has repeatedly been found (and this is evident in the following case reports) that intermittent syringe suction and siphonage into a bucket are, relative to continuous suction, of very low efficiency. Consequently, continuous suction is recommended as a routine measure as soon as the tube has been passed.

After consideration of the various methods for developing negative pressure, it was decided that the most suitable for alimentary suction was the readily available simple silent continuous gentle suction obtained from a three-bottle water-siphonage system evolved from Wangensteen's (1942). The apparatus described by Branch (1938), consisting of two bottles fitted in a reversible frame which is rotated through 180° when necessary, was used for a considerable time. Because of various difficulties with Branch's apparatus the apparatus to be described was evolved with two considerations in view: firstly, that it would require a minimum of attention and consequently a minimum of disturbance of the patient; and secondly, that its construction and method of management would be such as to be readily understood by the nursing staff.

The stand is constructed of wood and is standard equipment primarily intended for other ward purposes (Figure II). The top shelf is five feet and the lower three feet six inches above floor level. The bucket rests on the floor. The top bottle, holding three to six and a half gallons according to the size chosen, is primarily used as a container for industrial acids and other solutions, is known as a "carboy", and is obtainable through surgical suppliers, or from woollen mills or domestic gas companies. The receptacle on the middle shelf is a large milk bottle. The two corks are rubber, of appropriate size, and each has two holes. Tubes A, B, C, D and E are of copper, five-sixteenths of an inch in external diameter. Tube A projects one and a half inches further into the bottle than tube B, so that aspirated material emerging from A will not accidentally pass into B. Tube C projects just through the cork. Tube D is adjusted so that its lower end just clears the bottom of the bottle when the cork is firmly in position. Tube E is straight and merely rests in the bucket. A short piece of rubber pressure tubing is slipped over its lower end, of such length and diameter as to allow the tip of a large metal ear syringe to fit firmly when required. The lower end of this rubber tip is notched so that it cannot be occluded by the bottom of the bucket.

A length of pressure rubber tubing (tube 3) connects one limb of an inverted glass Y-tube (F) to the suction side of the particular indwelling tube in use. The second limb of tube F is connected to a short length of non-pressure rubber tubing (tube 2) carrying screw clip W, which in turn is connected by the tapered glass connexion G to an eight-inch length of rubber tubing (tube 1) of three-sixteenths of an inch outside diameter (for example, that used on tension sutures). The stem of the Y-tube is

connected to the trap bottle as shown, the Y-tube being firmly attached to the stand by adhesive strapping at the height shown and in such a way that the glass of the tube remains reasonably uncovered. Screw clip X is placed just above the Y-tube, and the rubber tubing on each side of the clip is firmly strapped to the stand.

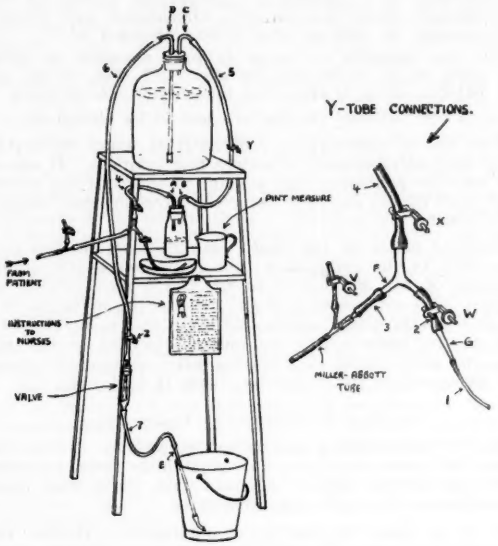


FIGURE II.

The writer's modification of Wangensteen's suction apparatus.

Tubes 4, 5, 6 and 7 are rubber pressure tubing of three-eighths of an inch outside diameter.

The valve, which is firmly fixed by strapping to the stand at as low a level as practicable, is shown in detail in Figure III, the essential feature being a rubber condom with the tip of the teat cut off, slipped onto the glass tube and tied in position with silk. The condom, with just sufficient cut off its lower end, hangs suspended loosely in a glass urethral syringe barrel.

Screw clips (Figure III) are placed on the tubing at points V, W, X, Y and Z, the type shown ensuring positive closure without damage to the tube.

Resting on the middle shelf is a pint measure containing sterile saline, a sterile kidney dish containing a ten-millilitre "Record" syringe and the lower end of tube 1, a metal ear syringe, and a mounted copy of "Instructions to Nurses" as described later.

Mode of Use.

With the apparatus set up as in Figure II, and all screw clips except Z closed, the cork is removed from the top bottle and rested on the lip of the opening with the tubes C and D straddling the lip, the longer still projecting into the bottle. This allows adequate room to fill the bottle almost to the top with tap water. The cork is firmly replaced, tube E is held in the left hand, the nozzle of the metal ear syringe, with the plunger pushed in, is inserted into the rubber tip of E, the plunger is quickly withdrawn, the syringe is removed, the tube E is placed in the bottle. Siphonage into the bucket is thus initiated, usually with one suck of the syringe. Tube 3 being attached to the indwelling tube, and the cork in the bottle of the middle shelf firmly inserted, clips X and Y are released. A flow of alimentary fluid contents alternating with gas then commences and its progress is readily observed in the Y-tube.

Five features of construction merit comment.

1. The by-pass ending in the narrow tube resting in the kidney dish allows ready syringing through of the indwelling tube if it becomes blocked. Clip X is closed. The "Record" syringe, charged with saline from the jug, is slipped on to tube 1 (the diameter of which is such that the end firmly grasps the syringe), clip W is opened and the indwelling tube is syringed until clear. Clip W is closed, the syringe is disconnected, and clip X is opened. Resumption of suction flow is observed in the Y-tube. It is in the early stages, when the aspirated fluid is gastric and contains flocculent material and mucus, that blockage is most likely to occur, and frequent observation of the Y-tube is necessary. When the Miller-Abbott balloon is in the duodenum and small bowel the fluid becomes thinner and more homogeneous.

It is an advantage to use an old artery forceps or clamp in place of clips X and W. As at any one time one tube is always patent, the clamp is transferred back and forth from tube to tube as required.

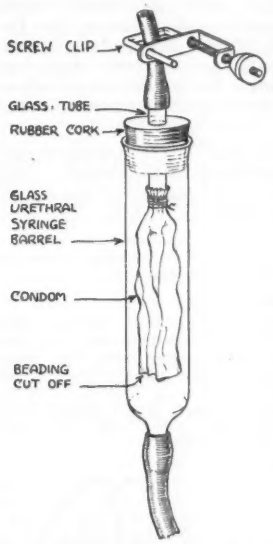


FIGURE III.

The valve portion of the suction apparatus.

2. The surprisingly large reservoir (top bottle) in the form of a carboy was chosen because of the rapid emptying of this bottle in the early stages of alimentary decompression, owing not only to the large amounts of fluid aspirated, but also to the often considerable amounts of intestinal gas. A small bottle was found to demand an excessive amount of watching and refilling, which was not only irksome to the nursing staff, but disturbed the patient and often his neighbours in the middle of the night. It is found that if the carboy is filled as the patient settles for the night, it usually requires no further attention until morning. After the first day of suction the quantity of gas aspirated becomes much less and attention to the top bottle is required only at long intervals. In several cases the interval was three to five days.

3. Before the valve (Figure III) was incorporated, it was frequently found on visiting the patient that suction had ceased owing to disappearance of water from tube 6, notwithstanding that the fluid level in the carboy was above the lower end of tube D. It was some time before the explanation of this phenomenon was discovered. The bucket has a much smaller capacity than the carboy, and therefore in the early stages has to be emptied frequently. Not uncommonly, when the bucket is nearly full, a state of equilibrium becomes established in which a considerable degree of negative pressure develops in the carboy, which is balanced by the column of water in tube 6 and the column of fluid in the bucket above the lower end of tube E. If the bucket is then emptied, the column of fluid in E and 6 is sucked up into the carboy, and after it, with very considerable gurgling, a large quantity of air, with resultant loss of a good "head" of negative pressure and loss of the siphon water column, which often is not detected for some time. Merely to clip the lower end of tube 6 before removing it from the bucket and not to open it until the end of E has been placed in a bucket with a few inches of water in the bottom, will not prevent this regurgitation if the state of equilibrium is in existence at the time when the full bucket is emptied. It was found that the remedy was to insert a valve in column 6 E well below the bottom of the carboy, which would close as soon as some retrograde movement of fluid occurred. With such a valve, the bucket could be emptied with preservation of the siphon column. The tendency to suck back

into the carboy is seen, by observation of the valve, to persist until further flow of fluid and gas from the alimentary tract occurs to such a degree as to lower the negative pressure in the carboy enough to allow fluid to run into the bucket again. The use of a condom for the valve was suggested by its use as a Miller-Abbott balloon. It was found to be remarkably efficient. There is a tendency for leakage to occur where the condom is tied to the glass tube. It may need encirclement with strapping to prevent this. In each new case a fresh condom should be used, as this deteriorates during storage after having been used. The temptation to use the more readily available glove finger or finger cot must be resisted, as neither is satisfactory.

4. The rigid tube E eliminates the irritating tendency of rubber tubing to curl up in the wrong direction.

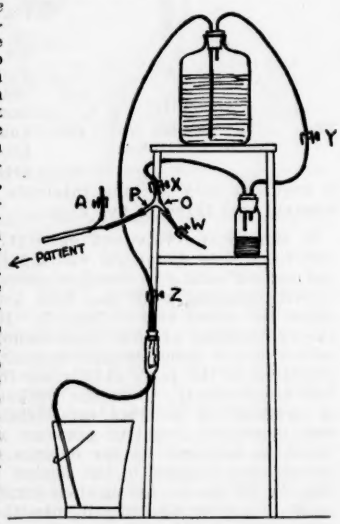
5. Pressure rubber tubing must be used where described to avoid interference with suction by the collapse that occurs with thin-walled tubing. It is preferable for all rubber parts to be disconnected after use, and the copper tubes to be pulled out of the corks; otherwise air leaks are likely to develop at metal-rubber and glass-rubber junctions.

Many practical points in the use of the apparatus are described in the following "instructions to nurses". This list of instructions is attached to the stand and the nurses are asked to make themselves familiar with it.

MANAGEMENT OF SUCTION APPARATUS AND MILLER-ABBOTT TUBE.

Watch glass Y-tube.

If the flow has stopped: (1) Make certain pins fastening tube to pillow are not compressing the tube and that tube is not under the patient's head. (2) Loosen clip "W": (*a*) A flow of bubbles up the Y-tube proves a negative pressure is present. If so, close clip "W" and syringe through Miller-Abbott tube (see below). (*b*) If no bubbles, close clip "W", and restore flow into bucket (see below).



To syringe through Miller-Abbott tube, close clip "X", open clip "W", attach syringe, filled with saline from pint measure on middle shelf, to tube in kidney dish (attached to "O"), and inject saline. Alternating aspiration and injection may be necessary to clear the tube. When clear, close "W" and open "X".

To restore flow into bucket, insert ear syringe into rubber tip of tube in bucket, give one forcible suck, remove syringe, and replace tube in bucket. If water fails to flow, repeat suction.

If top bottle is emptying rapidly, there is a leak in the system. (1) Observe glass Y-tube; if there is a flow of small bubbles up limb "P", inspect for air leak the attachment of the Miller-Abbott tubing to its metal Y connection, if there is a flow of small bubbles up limb "O" tighten screw "W". (2) See that corks of both bottles are fitting firmly. Rapid emptying, to establish a good negative pressure, is to be expected for a short period after filling top bottle. (3) Replace perished rubber tubing or corks.

To fill top bottle, close clips "Y" and "Z", remove cork and rest it on lip of bottle neck with short metal tube on outside of lip and long tube on inside. Fill bottle not quite to top. Replace cork firmly, open clip "Z", and if water fails to flow into bucket, restore flow (see above). When water is running into bucket open clip "Y".

To empty bucket, close clip "Z", transfer tube to an empty bucket and re-open clip.

To empty bottle on middle shelf, tightly close clip "Y", remove cork, empty bottle, replace cork and re-open "Y". Record in appropriate column of fluid balance chart the quantity, appearance and smell of contents as well as time it was disposed of.

The quantity of saline in pint measure on middle shelf is to be entered on the "IN" side of the fluid balance chart at time pint measure is placed there.

Tube "A" and its clip are *not* to be disturbed.

This list of instructions evolved from many attempts to train successive groups of sisters and trainees. It appears to cover the problems that are apt to confront the nursing staff from day to day, and saves the surgeon many unnecessary visits.

Another cause of too rapid emptying of the top bottle is a leak in the indwelling tube indicated by the audible passage of air without fluid up the Y-tube, which may persist with compression of the tube at the external nares. If this compression causes air flow to cease, the lower end of the balloon may be found impacted in the nasopharynx as a result of vomiting after apparently successful passage into the stomach (Cases II and VII).

SUCTION IN INTESTINAL OBSTRUCTION.

The term intestinal obstruction being used in its widest sense to include paralytic and peritonitic ileus, experience with intubation suction suggests that it is best treated according to the following principles.

1. In all cases of small-bowel obstruction, whether early or late, intubation suction should be commenced without delay, the necessity for operative treatment in addition depending, commonly in this order, on (i) the presence of strangulation, (ii) the failure of attainment of satisfactory decompression, (iii) inability to maintain adequate nutrition, and (iv) the failure of resolution of the cause of the obstruction. The one exception is obstruction from external hernia, in which the patient's condition is such that operation appears likely to result in uneventful recovery. Suction is then used only if indications arise later.

2. If a large-bowel obstruction is situated at the ileo-caecal valve, Miller-Abbott suction is used as in small-bowel obstructions. If the obstruction lies distal thereto, and if proximal decompression is required, this should be provided by intubation only when free regurgitation through an incompetent ileo-caecal valve is present.

Small-Bowel Obstruction.

The four factors indicating operation require consideration.

The Strangulation Factor.

When strangulation is obviously present, a few hours' pre-operative treatment is allowed, consisting of the intravenous correction of dehydration and salt depletion and the administration of serum or blood, together with the institution of suction decompression via a Miller-Abbott tube. Apart from the decompression's preventing regurgitation of vomitus during anæsthesia as well as making operation technically easier, the gradual nature of its establishment (even though this should be incomplete at operation) greatly diminishes the risks (Gius and Peterson, 1944; Knight, 1937) associated with the sudden decompression inevitably accompanying laparotomy. Suction is continued during and for some days after operation.

The suction should be continuous (the suction apparatus being taken to the operating theatre) from the time of intubation to the time when suction is no longer necessary. Notwithstanding the suggestion of others (Rodney Smith, 1948; Devine, 1946) that a gastric or duodenal tube should be given a trial, the writer prefers

to use a Miller-Abbott tube from the start, using a short tube as a preliminary only when a meal has recently been taken and only until the particulate matter has been evacuated.

The value of long-tube suction as an adjunct to early operation in cases of strangulated femoral hernia is suggested by the findings of Jarboe and Pratt (1947). They review 104 cases at the Mayo Clinic up to 1946, in which long-tube suction apparently formed no part of the treatment. The mortality rate of resection of small bowel was 62·5%. Of these fatal cases, in which end-to-end anastomosis was performed, post-mortem examination in half revealed "almost complete obstruction at the site of the anastomosis, in addition to leakage through the gaping suture holes in the peritoneum". The value of decompression of the bowel above the anastomosis is therefore suggested; but what were the results in which this was provided for by enterostomy? In seven such cases, five of the patients died. Of the fatal cases, necrosis and sloughing of the wall of the bowel around the tube with leakage of much foul fluid in the peritoneal cavity resulted in two deaths, and in another case "the ileum was obstructed completely at the site of the enterostomy". In another seven cases, enterostomy proximal to doubtfully viable bowel which was returned to the abdomen resulted in five deaths. Of these fourteen cases, necropsy in ten revealed "necrosis of that portion of the bowel around the tube, with leakage of foul fluid into the peritoneal cavity in nearly all cases. In two cases marked constriction about the enterostomy tube had caused partial intestinal obstruction". Jarboe and Pratt therefore come to the conclusion, obvious to those experienced in long-tube suction, that in the above-described circumstances suction is infinitely superior to enterostomy as a means of providing decompression.

Gatch and Montgomery (1945) paint a similar gloomy picture of the treatment of strangulated hernia containing gangrenous bowel, in which long-tube suction had not formed part of the treatment. They state that "methods which require an enterostomy are unsatisfactory and dangerous". When gangrenous bowel is present they advocate (i) incision of the hernial sac together with the gangrenous bowel within it, (ii) deflation of the obstructed bowel down to the hernial ring with a Miller-Abbott tube, and (iii) laparotomy, after the tube has been in place for about a week, with excision of the affected bowel followed by immediate intraabdominal anastomosis and post-operative protection of this suture line by the Miller-Abbott tube. They indicate that in all cases the peritoneal cavity remained shut off from the exteriorized bowel by firm attachment of the living bowel to the femoral ring.

Bodenheimer and others (1947) regard enterostomy as inferior to suction drainage, and in their recent cases used it only when the latter had failed—"usually multiple obstructions or inflammatory processes in which it was impossible completely to free the bowel". In their conclusions they state: "We are endeavouring to eliminate enterostomy as a definitive treatment so far as possible."

Enterostomy should not be considered, as it is by these writers, as competitive or alternative to intubation. It is to be regarded as complementary—as a valuable method of treatment to be used in conjunction with intubation according to definite indications discussed later.

When strangulation is absent, suction is continued without arbitrary time limit, the appearance of features suggesting strangulation constituting an indication for operation.

Conflict of opinion concerning the advisability of using prolonged intubation decompression in intestinal obstruction emanates from the fear of the possible consequences of strangulation. Of a total of three deaths in the writer's series, two resulted from internal strangulation of small bowel under a band. The method is to be used subject to two distinct conceptions in regard to strangulation. The first is that embarrassment of the circulation of the bowel in obstruction results from two separate mechanisms: (*a*) That in which distension causes occlusion of the vessels in the wall of the bowel by direct stretching, and (*b*) that in which occlusion of the mesenteric vessels results from thrombosis, or from torsion, encirclement or invagination of the bowel.

The second type, the extramural, is that commonly taken to be meant by the term "strangulation". It is from this type that disaster results from the misuse of suction. The unqualified word "strangulation", as used herein, is to be taken as meaning extramural strangulation.

The first type, the intramural type, occurs to varying degree in all obstructions. It constitutes the main danger of closed-loop obstruction particularly of the colon, but it is also important in small-bowel obstruction other than closed-loop obstruction, in that while it may evoke clinical features suggesting extramural strangulation, it is always relieved by successful suction intubation. Extramural strangulation of mild degree may be relieved by suction decompression (as indicated in quoted cases), but this cannot and should not be relied on.

The importance of intramural strangulation is to be emphasized. Knight (1937), though writing primarily on the effects of extramural strangulation, states that "we have observed depressor reactions in the peritoneal fluid in late cases of simple obstruction with marked distension but without (extramural) strangulation". He quotes Taylor, Wild and Harrison's findings of experimental distension without obstruction of animal's intestine causing death "with all the symptoms of experimental strangulation". He also makes the following statement: "In cases where exteriorization is performed, avoidance of distension in the upper segment is of importance, since it appears that such distension results in the appearance of further depressor substances in the peritoneal fluid."

If these conclusions are correct, a period of intubation decompression before operation, whether for extramural strangulation or simple obstruction, is of considerable value, not only because it renders operation technically easier and allows time for correction of biochemical deficiencies, but also because it gradually relieves intramural strangulation with a consequent fall in the production and absorption of toxic depressor substances.

If extramural strangulation is present, toxic substances are liberated suddenly and in quantity when the obstruction is relieved surgically. The effect of these is minimized if the pre-operative production of similar substances from the proximal distended portion of the bowel has been diminished by preliminary intubation decompression. Continuance of the intubation decompression into the post-operative period is similarly indicated.

The second conception is that extramural strangulation is a condition which may exist, when the patient is first examined, in various grades of severity, and which, during the period of conservative decompression, may not only become more pronounced, but may resolve as decompression becomes established.

That strangulation frequently progresses to a degree sufficient to cause peritoneal damage and then spontaneously resolves is evidenced by the old adhesions commonly found between bowel loops in cases of long-standing hernia. In Case II (Figure VII) multiple old adhesions were found between all small bowel loops that had passed through an internal hernial orifice, no adhesions being present in any other bowel coils. In Case III similar adhesions were present between all coils, distal to the main obstruction, where these coils passed beneath the transverse taut adhesive band (Figure IX).

It is unfortunate that there is a tendency to regard intestinal obstruction as being either mechanical or functional, with the implication that the division is clear-cut. Intubation decompression can be used intelligently only if all patients are considered, as emphasized by Crowley and Johnston (1941), as suffering from interruption of the normal flow of intestinal contents as the result of the existence of varying proportions of (*a*) mechanical occlusion of the intestinal lumen, (*b*) reduction of the peristalsis by disturbance of the nervous control or muscular efficiency or both, and (*c*) embarrassment of the circulation to the bowel (that is, strangulation) to such a degree as to cause (*a*) or (*b*) or both. Each factor, if allowed to operate for long enough, tends to cause the development of the others.

A combination of the three acting simultaneously might be sufficient to produce complete obstruction, whereas each acting alone might be insufficient. These conceptions are the basis of the use of prolonged intubation decompression in obstruction. It is used with the idea that if one or more of the factors can be relieved, then others including strangulation may spontaneously resolve.

Removal of the fluid and gas contents of the bowel above the site of obstruction, among other things, allows the bowel to collapse and become flaccid. This in itself relieves the varying degree of intramural strangulation which always accompanies distension. In addition it may result in decrease in tension of an adhesive band (Cases IV and V), with resultant decrease in a possible compressive or kinking action of the band on adjacent bowel and its vascular supply. It will facilitate unfolding of the kinks and twists often found in tense distended bowel, which each constitute a mechanical block. The generalized decrease in intraabdominal pressure results in a general improvement in the circulation of all abdominal organs. All these factors materially facilitate natural subsidence of other pathological changes contributing to the cause or resulting from the obstruction. The principle underlying the cure of recent adhesive post-operative obstruction is first to establish decompression and then to maintain it for sufficient time to allow inflammatory congestion and œdema to subside and plastic adhesions to absorb.

The suspicion of the existence of a minor degree of circulatory embarrassment should therefore not indicate precipitate laparotomy. A short trial of suction decompression may result in disappearance of all features suggestive of strangulation. However, if during conservative decompression subsidence of features suggesting strangulation is not prompt or if evidence of increase in the degree of strangulation occurs, or if signs of strangulation appear in a patient initially regarded as not suffering from strangulation, then operation should be performed without further delay, but the tube left in position.

The problem is not to prove that extramural strangulation is present, but to satisfy oneself that it is not. This is not made easier by the fact that the basic pathological changes and therefore some of the clinical manifestations resulting from intramural strangulation are essentially similar to those of extramural strangulation.

It has also to be remembered that the clinical features of extramural strangulation do not necessarily become more apparent in a gradual and progressive manner. They may remain more or less stationary for some days, possibly with some general improvement as a result of decompression of proximal loops, only to develop dramatically into those of peripheral circulatory failure with death within a matter of hours (Case VI).

It must therefore be stated that the only safe procedure is to operate (after a short period of preliminary suction) in any case in which a non-resolving extramural strangulation cannot be excluded.

The presence of extramural strangulation is suggested by the following signs.

1. Vomiting that appears at the onset of the illness, is violent in nature and is persistent. The vomiting of a simple obstruction commonly appears relatively late, is gently regurgitant rather than violent and ceases as soon as intubation suction is commenced.

2. Shock, with evidence of peripheral circulatory failure, that appears at the onset of the illness and persists in spite of apparently satisfactory intubation decompression and intravenous therapy. A striking feature in simple obstruction is the rapid and permanent recovery from the peripheral circulatory failure associated with the dehydration of marked distension and vomiting that follows intubation suction and intravenous replacement therapy.

3. Pain that is continuously present. Although attacks of colicky pain may recur, some pain persists between the attacks. Complete absence of pain between attacks of colic suggests that strangulation is not present.

4. A localized area of tenderness (direct and rebound) and perhaps rigidity. This suggests an underlying strangulation, especially if the area should gradually become more extensive. Generalized tenderness is present at times in a non-strangulating obstruction, but such tenderness should be regarded as suggestive of at least intramural strangulation.

5. A palpable mass or a localized area of persisting or increasing distension (Case V) corresponding to the area of tenderness. This is suggestive, as is a tender mass palpable *per rectum*. Absence of local tenderness, rigidity or mass does not exclude strangulation.

6. An increase in temperature, pulse rate and white blood cell count, which is said to take place with the duration and severity of strangulation. Usually in a purely mechanical obstruction the temperature and pulse rate remain normal; but when these patients are having continuous suction they are also necessarily having continuous intravenous administration of fluid containing glucose. This latter procedure usually results after a few days in thrombophlebitis causing fever often without noticeably raised pulse rate. Whether these features are due to phlebitis or to strangulation must therefore be decided, the occurrence of a normal pulse rate at some time during the febrile period being in favour of the former (Case II). However, neither normal temperature nor normal pulse rate necessarily excludes extramural strangulation. In Case VI, a temperature of 98° F. and a pulse rate of 72 per minute (the last charted) were recorded nine hours before the discovery of steadily progressive peripheral circulatory failure, which resulted in death ten hours later from internal strangulation of a bowel loop. The four-hourly pulse rates for the preceding thirty-six hours were 96, 76, 84, 72, 76 and 72 per minute, a temperature (99° F.) above normal being recorded once. Wangensteen, Mason and Zintel, and others, find that variations in the white cell count are not characteristic. A rising count suggests strangulation, but a normal count dots not exclude it.

7. The radiological appearances in small-bowel obstruction varying according to the presence or absence of a closed loop. At one extreme, simple obstruction from a single non-strangulating agent at or near the ileo-caecal valve, in which distension is well advanced, gives the typical step-ladder arrangement of distended coils passing transversely. At the other extreme is a closed-loop obstruction of wide extent, in which the proximal block is situated high in the jejunum, the loop is much distended, but as yet distension above the proximal block is minimal. In this case the step-ladder arrangement is absent, the loop being irregularly disposed in the abdomen, its degree of distension being considerable.

Between these extremes variable pictures occur. In the early stages of a single simple block a few coils only may appear distended in the skiagram, and the condition may at first appear to be localized. This appearance may be indistinguishable from that of an early closed-loop obstruction. In a closed-loop obstruction of very short length, especially if it involves the lower terminal portion of the ileum, the gaseous distension of the loop may remain slight, and distension of the bowel above the upper block may give a step-ladder appearance. Such appearances will suggest a simple obstruction.

The radiological appearances therefore cannot be relied on to exclude extramural strangulation, although some appearances will justify a presumptive diagnosis of a closed-loop obstruction. Thus, on the one hand, an isolated loop of distended small bowel in the presence of other suggestive clinical features is almost pathognomonic. This is likely to be seen early in the illness. On the other hand, if distension of the closed loop is slight but that above the upper block is pronounced, decompression of the proximal part of the bowel by intubation usually soon reveals the radiological appearances suggestive of a closed loop (Cases I, V and VII; Figures IV and V), except when the loop contains fluid only (Case VIII).

In addition to those mentioned above, clinical features characteristic of each of the recognized types of strangulation may be present.

It follows from the above that the writer does not necessarily perform early operation on patients whose history of obstruction is of less than forty-eight hours' duration, as McKittrick (1948), Chesterman (1945), and Mason and Zintel (1946) suggest should be done as routine, unless the factor of extramural strangulation suggests that early operation is advisable.

One reason for early operation suggested by these writers is that it will be performed before distension is sufficiently advanced to make the procedure difficult.

There is apt to be considerable discrepancy between clinical radiological and operative evidence of distension. Gius and Peterson (1944) make the following statement:

> Distension may be a serious complicating factor in obstruction before it is clinically appreciable. We have noted that frequently only a single loop or several small loops of distended bowel may be evident on the survey film, yet distension out of all proportion to that expected is encountered at operation.

On the other hand, it is not uncommon to find an X-ray film show much distended small bowel when the abdomen on clinical examination is not obviously distended, and in the same case to find at operation not only that troublesome prolapse of distended coils occurs, but that the release of the external supporting pressure afforded by the unopened abdominal wall allows a distended coil to become further distended to such a degree that multiple splitting of its serosa occurs.

In these cases in which the history is short, intubation suction is likely to result in rapid decompression; at this stage operation will probably be performed, except when the abnormality causing obstruction is expected to resolve, or when, because of special features, it is thought better to defer operation. In other words, early operation is likely to be performed, but not merely because the history is shorter than some arbitrary period of time.

The Failure of Attainment of Satisfactory Decompression.

In the writer's series, Miller-Abbott intubation has satisfactorily decompressed all small bowel obstructions except those in which closed-loop obstruction has been present.

When the latter is associated with extramural strangulation (as, for example, in strangulated internal hernia and volvulus), the necessity for operation usually becomes apparent early, and operation is performed irrespective of the degree of decompression of the proximal part of the bowel, and with the tube *in situ*.

Where extramural strangulation is absent, as for example when adhesive obstruction occurs at two levels in the bowel (Cases VII and VIII), the sequence of events is commonly as follows:

The Miller-Abbott tube produces early and satisfactory decompression of the bowel above the most proximal obstruction. When this has been attained, the presence of the closed-loop obstruction is suggested in serial skiagrams (Figures IV and V) by the persistence of one or more loops of distension which maintain a constant position in successive films, together with the assumption by the tube tip of a fairly fixed position in the abdomen (usually away from the ileo-caecal angle) after only partial progression down the bowel, a portion of the abdomen frequently remaining persistently free of loops of tubing. At the same time there occurs recurrent colic synchronous with borborygmi, localized distension and perhaps tenderness in the area of this loop (Cases I, V, VII and VIII).

At this stage, if it is thought advisable, suction decompression may be maintained in the hope that the obstruction may relent at one or both ends of the closed loop. Such resolution is suggested by diminution of the degree of distension in the loop as seen in daily skiagrams (Case VII; Figure V), and by lessening of the colic, borborygmi and local distension. In addition, reopening of the lumen at the distal obstruction is suggested by the appearance of gas in the colon in the serial skiagrams (Figure IV) and the passage of flatus and perhaps fæces *per anum*, while reopening of the lumen at the proximal obstruction is suggested by alteration of the aspirated fluid from the greenish-brown fluid coming away in a steady volume characteristic of the decompressed loop, to fæculent fluid coming away in irregular gushes.

In the early stages of resolution of any obstruction the lumen is patent only intermittently. Such a state may persist without further resolution (Case VII). In favourable cases the obstructed areas become continuously patent. However, if repeated skiagrams reveal an increase in the diameter of a distended loop, and if none of the above-mentioned changes suggestive of reopening appear, operation is indicated (Cases I, II and V; Figure IV).

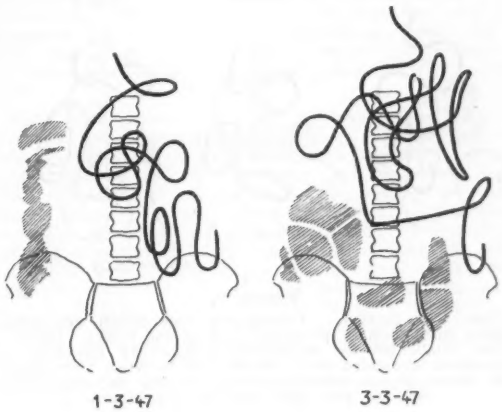


FIGURE IV.

Case I (M.F.). Closed-loop obstruction requiring enterostomy because of increasing distension of the loop. Dense old adhesions of small bowel to pelvic viscera were present.

This conception, that the existence of a closed-loop obstruction is the main if not the sole cause of failure to attain satisfactory decompression by intubation, does not appear to have been clearly indicated previously.

It is the writer's impression that in those cases in which others have operated because of unsatisfactory tube decompression, the reason for failure to obtain decompression was not failure of the tube to pass into the duodenum, as was usually suggested, but the presence of a closed-loop obstruction.

Intubation at the best can decompress the bowel only above the most proximal block, and this is true whether the tube is swallowed, is passed through a gastrostomy opening (Rodney Smith, 1948), or is passed through a high jejunostomy opening (Devine, 1946).

The writer is not convinced that, provided the management described later is used, indication should ever exist for Rodney Smith's "intestinal suction gastrostomy", this being the passage through the pylorus of the Miller-Abbott tube by way of a gastrostomy.

After a trial period of suction, operation may be indicated because of failure of attainment of decompression; according to the patient's condition and the abnormality found, such operation should consist of either direct attack on the obstruction (Cases II, V, VII and VIII), of or enterostomy into the closed loop (Case I).

The post-operative treatment in each case should include intubation decompression of the proximal part of the bowel. If the obstruction has been directly attacked, intubation will prevent post-operative ileus. If enterostomy into a closed loop has been performed, such a procedure will have no effect on the bowel above the proximal block unless decompression of the loop facilitates early resolution of that block. Therefore this bowel will still need intubation decompression.

According to this view, enterostomy is likely to fail to produce satisfactory decompression in the following circumstances: if it is not combined with decompression of the bowel above the proximal block; if it has in error

been performed on the bowel above the proximal block; if it has been performed on a closed loop and other closed loops are present; or if it has been performed on a very long closed loop, in which multiple kinks of the "bent bike-tube" type are present and the paralytic factor is pronounced.

It is in this last-mentioned type that Miller-Abbott intubation through a jejunostomy opening (Devine, 1946) distal to the upper block may prove of advantage if combined with intubation of the bowel above.

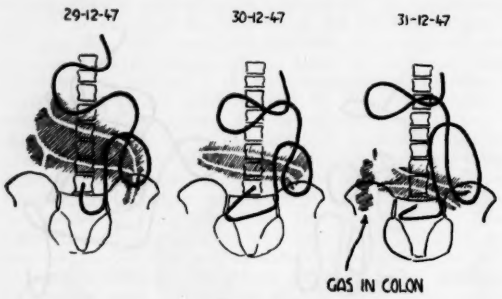


FIGURE V.

Case VII (E.W.). Closed-loop obstruction resolving after intubation of proximal part of the bowel. Operation (indicated by nutritional difficulties) disclosed a partially distended long closed loop between two areas of recent plastic adhesions and complete decompression of the proximal part of the bowel.

This conception places intubation and enterostomy in their correct relationship as complementary methods of treatment. One without the other may prove inadequate.

Enterostomy as a method of treatment of intestinal obstruction has probably proved of variable value in the past, not because it was essentially a poor method, but because it was not appreciated that it was often necessary to combine enterostomy into a closed loop with decompression of the bowel above the proximal block of that loop.

CASE I.—Miss M.F., aged sixteen years, on February 13, 1947, had a pelvic appendix with an acutely inflamed tip removed through a McBurney's incision. For the next ten days her temperature and pulse rate were elevated, and she complained of abdominal pain and "wind" and vomited small amounts frequently. On the third post-operative day an enema gave a good result. On the fifth day her bowels were open frequently. On the seventh day an enema gave a good result. On the eighth day her abdomen was distended, but she had a good bowel action. The temperature and pulse rate then fell and her symptoms abated.

On February 26 her temperature and pulse rate rose again, and abdominal pain, nausea and constipation recurred. Her abdomen was distended but not tender. Two enemata gave negative results.

Next day (February 27) her symptoms were unchanged, and an X-ray film revealed much small-bowel distension with multiple fluid levels. At 7.30 p.m. a Miller-Abbott tube was passed and continuous suction was applied. Seven and a half hours later the aspirated fluid became greenish-brown and started to run freely, this suggesting that the tube tip had passed through the pylorus.

On February 28 an X-ray film taken at 9 a.m. showed the tube tip at the duodeno-jejunal flexure. The balloon was then partly filled with water.

On March 1 an X-ray film taken at 11.45 a.m. showed the tube tip well down the small bowel; another film at 6 p.m. showed considerable further tube migration (Figure IV).

In spite of this rapid progression of the tube, of the aspiration of large quantities of fæculant fluid, of some decrease in abdominal distension and of the passage of some flatus *per rectum*, some distension and abdominal pain persisted.

On March 3 an enema resulted in the passage of flatus only. An X-ray film taken at 11 a.m. showed the Miller-Abbott tube to be confined to the upper and left portions of the abdomen, there being some distended bowel loops in the lower right quadrant of the abdomen. A film taken six hours later showed no evidence of decompression of these coils (Figure IV). During the night the patient vomited 12 ounces of yellow fluid. A diagnosis of residual closed-loop obstruction was made.

Operation on March 4 revealed extensive subacutely inflamed multiple adhesions between several distended coils of small bowel and the back of the body of the uterus and adnexæ. One coil contained the tip of the Miller-Abbott tube. As the latter appeared to be blocked, this loop was opened and the lumen of the tube was cleared. This enterotomy did not result in further decompression, and the incision was closed. It was found impossible to relieve the closed-loop obstruction by separation of the adhesions. During the attempt, an abscess cavity containing thick foul pus was opened into the back of the uterus. An enterostomy opening was made into the most distended loop, a drain tube was passed to the bottom of the abscess cavity and the abdomen was closed. Suction through the Miller-Abbott tube was continued without interruption.

The drain tube drained profusely for several days, the patient's temperature and pulse rate gradually falling to normal. Some purulent discharge was still present at the time of the catastrophe on March 25.

The enterostomy tube drained poorly and erratically for four days. After it had been cleared by the injection of saline, it drained freely for ten days (until March 19). It thereafter drained poorly.

On March 23, the enterostomy tube came out and was reinserted by the house surgeon; it did not drain at all.

The Miller-Abbott suction tube continued to drain well after operation. On March 9, as the patient's bowels had been well open the previous day and the enterostomy tube was draining well, the Miller-Abbott tube was removed. Her bowels were well open several times four days after operation (March 8), but not again until March 16, when they opened three times, each time with the production of abdominal pain. They were open again on March 23 and 25.

On March 25 she had been afebrile for ten days and was feeling well. The enterostomy tube, which had not drained since its reinsertion three days previously, came out and was reinserted by the house surgeon. This procedure was quickly followed by abdominal pain, which in a few hours became generalized, the abdomen becoming generally tender, slightly rigid but not distended, and the temperature rising to 100·2° F. and the pulse rate to 160 per minute. The following morning the temperature was 102·8° F. and the pulse rate 150 per minute. Pronounced general peritonitis was present. Frequent copious vomiting occurred, which soon became fæculent. As the enterostomy tube had not drained since its reinsertion it was removed. The enterostomy wound did not again drain intestinal contents.

At mid-day on March 26, the patient was suffering severely from shock with an imperceptible pulse. Continuous intravenous therapy had been instituted on March 25. A Miller-Abbott tube was inserted through the nose and continuous suction was applied. It immediately drained freely and continued to do so until it was removed on April 1. She remained very ill for several days, but the peritonitis gradually resolved, the temperature returning to normal by lysis at the end of four weeks. A normal bowel action occurred on March 29 and on March 30. Several bowel actions occurred on March 31, after which the Miller-Abbott tube was clipped off for twelve hours. As no discomfort ensued the tube was then removed and the giving of a non-residue diet was commenced. The original drain wound, from which drainage had been only slight for some days, now commenced to discharge profusely offensive excoriating fæculant fluid. This discharge persisted for several days, and gradually ceased. For four weeks she vomited occasionally, usually partly digested food after meals. She experienced intermittent attacks of pain and alternately periods of constipation and short attacks of the frequent passage of loose stools with occasional normal stools. The drainage fluid soon changed from fæculent to purulent, and this was discharged irregularly and at times profusely for some weeks. The patient gradually became normal in all respects. X-ray screening on May 5, 1947, showed that the barium flowed freely into the caecum.

At December, 1947, she was well, had been so for several months, and had no symptoms suggestive of obstruction.

Miller-Abbott decompression of the bowel above the closed loop rapidly revealed the clinical and radiological feature of the latter.

Enterostomy of such loop (and drainage of the abscess cavity) combined with persistence with intubation allowed

gradual subsidence of the pelvic inflamatory swelling with eventual restoration of the alimentary stream.

The peritonitis ileus resulting from the trauma of reinsertion of the enterostomy tube was successfully decompressed by further Miller-Abbott intubation, resolution occuring without operative interference becoming necessary.

Failure of the tube tip to leave the stomach is not in itself an indication for operation. Quite early in one's experience of intubation arises the question of how necessary and how urgent it is for the balloon of the Miller-Abbott tube to pass into the duodenum and descend into the lower part of the small bowel. Satisfactory decompression has frequently been attained when the tube tip has never left the stomach, and lack of migration in itself should cause no immediate concern provided evidence of strangulation or closed-loop obstruction does not appear. However, if the obstruction should prove unrelenting and if intubation is persisted in, many inadequacies of gastric suction become manifest.

Initial apparent failure to attain decompression is not in itself an indication for operation. As was previously indicated, the writer is not convinced that operation should ever be performed primarily to allow the passage of a Miller-Abbott tube through either a gastrostomy or a jejunostomy above a closed loop.

As will be set out in detail later, the period during which suction decompression is applied is divisible into two distinct phases. The first is that of establishment of adequate decompression, and the second is that of maintenance of decompression. The features of each are characteristic. Arbitrary time limits should not be imposed for either stage. The criteria of attainment of adequate decompression and those of restoration of normal flow of intestinal contents (that is, relief of the obstruction) are also distinctive. Nevertheless attainment of decompression is commonly mistaken for resolution of the obstruction.

Inability to Maintain Nourishment.

If the obstruction involves a high level in the jejunum, inability to maintain nourishment may be the factor demanding operation.

If an adequate length of small intestine lies proximal to the obstruction, once such length has been intubated, nourishment can be adequately maintained by oral feeding (Cases II and III).

If the obstruction is high in the jejunum, the short length of the bowel available may be insufficient for the absorption of adequate nourishment even though it may be predigested. A sense of fullness resulting in nausea and perhaps vomiting is then apt to follow ingestion of small quantities of food normally palatable (Case VII).

Operation should consist either of direct treatment of the obstruction or of enterostomy below the obstruction. If there is no obstruction below this enterostomy opening it is used for feeding purposes. Intubation suction of the proximal portion of the bowel is continued without interruption. As long as the aspirated fluid is clear golden intestinal fluid, it is injected through the enterostomy opening along with appropriate jejunal feeds.

Failure of Resolution of the Cause of the Obstruction.

Satisfactory decompression of the bowel above the obstruction is usually attained. At this stage, the probability that the cause of the obstruction may resolve after a reasonable period of decompression is assessed, and if resolution is regarded as improbable, operation is performed with tube *in situ* (Cases II and III).

Large-Bowel Obstruction.

If a large-bowel obstruction is situated at the ileo-caecal valve, Miller-Abbott suction is used as in small-bowel obstruction and as a pre-operative measure.

Three types of obstruction distal to the ileo-caecal valve occur: (i) that in which distension of the proximal part of the colon is pronounced, the ileo-caecal valve is competent, and the small bowel is not distended; (ii) that in which distension of the colon is slight, the valve is competent, and the small bowel is not distended; (iii) that in which the valve is incompetent and both the colon and the small bowel are distended.

Intubation suction is contraindicated in the first type of obstruction. For the second intubation suction may provide adequate pre-operative decompression. This does not directly decompress the colon. It merely helps to prevent further distension by stopping the flow of ileal contents into the obstructed colon which otherwise would occur. In this way it may delay the development of a degree of distension demanding operative relief sufficiently long for a mild incomplete colonic obstruction to resolve (Hexworth, 1946). Miller-Abbott suction has also been suggested (Allen, Welch and Donaldson, 1947) for the third type—that is, the type in which incompetence of the ileo-caecal valve allows regurgitation into the small bowel.

When the ileo-caecal valve is competent, the colon proximal to the obstruction constitutes a closed loop, and distension is early, is associated with a tensely distended thin-walled caecum, and therefore requires prompt operative decompression. At the same time, small-bowel distension is usually absent. When the valve is incompetent the colon proximal to the obstruction is maintained partially decompressed by regurgitation into the ileum, and pronounced colonic distension does not occur. Symptoms sufficient to cause the patient to seek advice may not develop until the changes found at operation are pronounced dilatation of several feet of ileum, moderate dilatation of the colon proximal to the obstruction, fairly pronounced hypertrophy of both the ileal and proximal colon walls, and almost complete obliteration by the growth of the lumen at the site of the obstruction.

Here, because the wall of the caecum and colon is hypertrophied and the intraluminal pressure rise has been gradual and never reaches a high level, decompression is usually not urgently required. In these cases intestinal intubation decompression is worthy of trial. If the obstruction persists, the aspirated fluid, as the tube tip descends, changes gradually from the thin, fæculant fluid typical of small-bowel obstruction to the thick, truly fæcal fluid characteristic of a caecostomy. This fluid may prove too thick to run through the tube, and decompression may thus prove inadequate (Case IX). However, it is is worthy of trial for two reasons. The obstructed part of the colon may become patent during the establishment of small-bowel decompression; and secondly, if the obstruction should be in the right side of the colon, it may obviate the necessity for caecostomy (Case XI), a most undesirable preliminary to right colectomy, and also afford useful post-operative decompression after subsequent ileo-colostomy.

The clinical picture and radiological appearances in these cases may suggest a simple ileal obstruction, and intestinal intubation may be commenced on this assumption. The gradual change to the thicker fæcal fluid will then suggest the correct diagnosis.

Three Types of Intestinal Obstruction.

In accordance with the foregoing principles cases of intestinal obstruction fall into one of three groups.

Group I consists of small-bowel obstruction with strangulation, and all large-bowel obstructions, which are dealt with according to the usual surgical principles, intubation suction being considered only as an adjunct to pre-operative treatment.

Group II comprises those small-bowel obstructions in which intubation is used for prolonged periods, with expectation that in most cases the obstruction will resolve without operation. These obstructions come under two headings: (i) post-operative obstruction occurring in the early post-operative period; (ii) the obstructions accompanying peritonitis.

(i) Post-operative obstruction of the alimentary tract is usually non-strangulating, and takes the form of acute dilatation of the stomach, œdematous obstruction at an anastomotic stoma, or intestinal obstruction of varying causation which is commonly labelled "post-operative ileus". Intubation decompression is applied, with the expectation that in most cases, after a variable period of

decompression, permanent resolution of all pathological changes will result. When satisfactory decompression has been attained, the tube is retained *in situ*, for several days if necessary, to maintain the decompression until the obstructing mechanism resolves (Cases I and X). During maintenance of decompression, provided that the tube tip is beyond the duodeno-jejunal flexure, oral feeding with a non-residue diet is practicable. If spontaneous relief of the obstructing mechanism does not occur, the subsequent management is as in Group III. In the early stages of treatment it is sufficient merely to determine that an obstruction somewhere along the alimentary tract is present.

Acute dilatation of the stomach is rare, occurs early, and will not be seen in its fully developed form if vomiting occurs early and is treated promptly by continuous intubation suction.

Œdematous obstruction at an anastomotic stoma is most commonly seen at the efferent stoma after gastrectomy. The obstruction in most cases resolves after a period of continuous gastric suction, provided that factors encouraging persistence of the œdema—for example, hypoproteinæmia—are corrected.

Post-operative intestinal obstruction may be truly paralytic without any associated mechanical element. Much more commonly it is primarily mechanical, and due to filmy fibrinous adhesions resulting from direct abrasive trauma to the visceral peritoneum during operation. Probably such adhesions form after a great number of abdominal operations. In the majority of cases they are absorbed without the production of symptoms. Occasionally their distribution is such as to result in anchoring and kinking of bowel loops sufficiently pronounced to cause obstructive symptoms. If in such obstruction decompression is maintained it usually resolves after a few days, and the resolution is likely to be permanent.

A third type of post-operative intestinal obstruction is that in which the adhesive area is more definitely established—for example, in the wall of an abscess cavity, around a drain tube, or at an inadequately peritonealized operative area such as the cervical stump or the deep surface of an operative incision. Although intubation may result in eventual reestablishment of intestinal patency, the adhesion may persist, become organized and at a later date cause further obstruction.

It is in post-operative ileus in particular that intubation suction has proved its worth. If the viewpoint is adopted that vomiting occurring after the first post-operative day is due to alimentary obstruction until proved otherwise, and if continuous intubation suction is promptly instituted simultaneously with continuous intravenous therapy and prohibition of all oral intake, then well developed post-operative ileus will rarely be seen. Such a policy may at times savour of "using a steam hammer to crack a nut". A "wait-and-see" attitude for a few days or inadequate intermittent syringe suction has frequently been regretted, but never has early continuous intubation suction.

(ii) Localized peritonitis consists of an area of congestion, œdema and fibrinous adhesions affecting one or more loops of bowel. If this should cause obstruction, such is chiefly mechanical, there being but little paralytic element in the absence of gross distension, and usually no extramural strangulation. The treatment is as in post-operative adhesive obstruction, the main difference being that resolution is likely to be slower and may remain incomplete. Intubation decompression is usually readily attained, and provided this is maintained for a sufficient period, the inflammation often subsides with restoration of intestinal patency. Such an area of inflammation may contain a focus of infection requiring appropriate treatment (for example, appendicectomy), or an abscess which may require drainage before resolution of the inflammation and obstruction will occur. In a minority of cases, the bowel may be completely obstructed at two levels with the consequent formation of a closed loop, and if both of these obstructions persist in spite of intubation suction, enterostomy as well as proximal suction will become necessary (Case I).

The ileus of diffuse or general peritonitis is initially paralytic, but the development of the inflammatory reaction (congestion, œdema and fibrinous deposit) soon adds a mechanical factor. If the infection is brought under control the inflammation may become localized in one or more situations, at each of which varying degrees of narrowing of the bowel lumen may occur.

The patient at this stage suffers from multiple mechanical obstructions of varying degree, with an associated pronounced paralytic element varying at different levels, and resulting from the combined effects of distension and the direct action of bacterial toxæmia.

The effect of intubation suction in such a case is therefore unpredictable. It will always do some good, in that it will provide a varying degree of relief of the distension above the most proximal block. If the patient is fortunate, rapid resolution of the inflammation at all areas may occur, so that the mechanical blocks gradually disappear. At the same time, control of the infection may result in diminution of the bacterial toxæmia factor contributing to the paralytic element of the ileus. Complete resolution of the obstruction may thus occur. Again, the paralytic factor may never resolve, and death may result from the paralytic element in the distension and from bacterial toxæmia. At the other extreme, the initial paralytic ileus may resolve with or without intubation, and without the inflammatory reaction ever causing mechanical block.

The indication therefore is that the ileus of general peritonitis is to be combated by treatment of the infection and by the early institution of intestinal intubation, it being borne in mind that the latter may produce early and satisfactory decompression, may result in decompression only after an initial period of apparent failure or may never produce satisfactory decompression.

It has to be remembered also that enterostomy into one or more distended loops may have to be performed in addition, and that apparent resolution of the obstruction as a result of such combined efforts may, perhaps days later, be followed by recurrence of obstructive symptoms requiring resumption of intubation (Case I).

Group III consists of the remaining non-strangulating small-bowel obstructions. In this group, conservative suction through a Miller-Abbott tube is used, with the knowledge that at the end of a variable period of suction operation will probably be performed. It is used because of the striking contrast existing between the conditions found at the elective operation performed on the nourished patient with decompressed bowel and in a good state of hydration, and those encountered in the dehydrated and starved patient with a distended abdomen, subjected to the usual emergency operation. The contrast is so striking that it is believed that the advantages far outweigh the dangers of overlooking a serious strangulation.

A further important feature of suction decompression is that it produces gradual decompression in contrast to the sudden decompression, with its attendant dangers (Gius and Peterson, 1944), inevitably associated with laparotomy.

Conservative suction is persisted with so long as it appears to be establishing decompression, and so long as signs of strangulation or unrelieved closed-loop obstruction are not appearing. If adequate decompression is attained, the decision as to whether decompression is to be maintained in the hope that the obstruction will relent depends on circumstances.

If, as is usual, clinical evidence suggests that laparotomy is desirable irrespective of whether the obstruction will relent or not, then it is performed without further delay provided biochemical deficiencies have been satisfactorily corrected and any desirable investigations have been completed (Cases II, III, IV and V).

Operation is performed with the tube *in situ* and as an elective procedure. In Case IV it was comfortably performed under "Pentothal" anæsthesia. In every case the patient's general condition has been excellent, and in most the bowel has been collapsed and readily packed when required; the lesion has been readily accessible, with healthy circulation, and amenable to the most radical of treatment. Procedures have been carried out which, even if practicable at operation in the presence of unrelieved distension, would have been associated with post-operative periods fraught with danger (Cases II and VIII).

In the occasional case in which operation has to be performed in the presence of inadequate decompression, the suction apparatus is taken to the operating theatre and suction is maintained through the operation. It is commonly found that the operative manipulations and the unkinking of distended bowel accompanying its delivery outside the abdomen result in the aspiration of considerable amounts of gas and fluid; thus conditions are provided which approach those of operation on a bowel completely decompressed pre-operatively.

In all cases the Miller-Abbott tube is left *in situ* for a few days post-operatively (usually until the bowels have opened), and if it has sufficiently migrated at operation it is passed to the anastomosis (if any).

If, when adequate decompression has been attained, avoidance of operation is regarded as desirable—for example, from factors such as extreme obesity, cardiac insufficiency *et cetera*—then a trial period of maintenance of decompression is adopted. This attitude appears justifiable also in regard to those patients who have had one or more operations for adhesions and are known to have "matting" together by broad adhesions of multiple coils of bowel. Those who when examined have a complete obstruction either present or imminent, and who give a history of previous attacks of partial obstruction which have spontaneously resolved, appear to be best treated in this way. That is, after tube decompression has been attained, nutrition is maintained by a non-residue diet given orally (with or without fluid given parenterally), and suction is persisted in for several days. This may result in subsidence of œdema at the site of obstruction, or spontaneous unkinking may be rendered possible by the collapse of decompressed coils. In this way intestinal patency may be reestablished and an operation avoided which might have proved unsatisfactory in respect to the ensuring of permanent cure. Obstruction may recur, but further intubation can be carried out. If, in these latter cases, difficulty is experienced in establishing decompression, suction should be persisted with for several days, so long as evidence of strangulation or closed-loop obstruction does not appear. Grimson and Hodge (1944) write on similar lines and give an excellent description with characteristic case reports of this problem of multiple adhesive obstruction. However, one of the two deaths in the series directly attributable to conservative intubation occurred from an undiagnosed small strangulated knuckle of bowel in an obese woman who had had two operations for non-strangulating obstruction from old adhesions.

In all cases in this group, the Miller-Abbott tube's role as a means of preparation for operation is emphasized, the time allowable depending not so much on the efficiency of decompression as on the likelihood of a strangulation existing.

If properly applied intubation suction combined with adequate parenteral feeding does not result in satisfactory decompression, then no other non-operative measures are likely to succeed. It is only in obstructions in which a mechanical element is absent that drug treatment designed to increase bowel movements should be considered; in surgical cases such obstructions are rare. Drug treatment in the presence of a mechanical element in the obstruction is likely to do harm, and it is contended that clinical determination of the absence of a mechanical element is difficult to make with safety.

The Details of Management of Intubation in Intestinal Obstruction.

If vomiting has been copious and repeated and the vomitus consists of thin fæculent fluid, a long intestinal tube is passed.

If a meal has recently been taken and vomiting has been slight, a large-bore stomach tube (such as a Wangensteen tube) is passed and the stomach is aspirated and washed out. It is then replaced by a long intestinal tube.

A constructional fault in the Miller-Abbott tube may prove disastrous. Great care must be used in its preparation. It should be carefully inspected for evidence of perished rubber and for leaks, fluid being injected under pressure along each lumen. A new balloon (that is, a condom) should be used for each case. Care should be used in tying it in position to ensure that the binding is not so tight as to occlude the lumina, and that when distended it is of satisfactory shape. The balloon is moderately distended with water, the amount of fluid needed being noted. To ensure that it is water-tight, the rubber tube is occluded and the balloon is compressed. The balloon is then emptied.

Without local anæsthesia, but with the balloon completely collapsed and twisted like a closed umbrella, the Miller-Abbott tube smeared with water-soluble lubricant is passed, with a screwing motion, through the more patent nostril, the patient sitting almost erect. The patient swallows water, held ready in the mouth, as the tube is passed through the naso-pharynx. He is told that the procedure will be uncomfortable, but is urged to resist vomiting and rapidly to swallow more fluid as the tube is fed in the nose. Even if vomiting is precipitated by the passage of the tube, the procedure is persisted in. Such vomiting at times may be profuse, but it and the obvious distress of the patient are not allowed to deter one from insisting on the patient's cooperation until the tube is in the stomach.

The tube is slowly passed through the nose for 68 centimetres—that is, to seven centimetres short of the 75 centimetre mark—the patient simultaneously swallowing a little fluid. The tube is fixed to the upper lip or nose by adhesive strapping. To minimize the tendency to coiling in the fundus, the tube is not allowed to enter the nose past this point until the tip is proved radiologically to be in the duodenum. Failure to ensure this is the most common cause of failure of the tube-tip to pass through the pylorus.

Continuous suction is applied with the patient propped up and inclined forward over a cardiac table, and the passage of the tube tip into the duodenum is awaited. This is indicated by two observations.

1. The fluid aspirated is inspected as it passes through the glass Y-tube (Figure II). When obstruction is present, one of two sequences occur. (i) The fluid may be at first colourless, grey or bile-stained gastric content, containing frothy mucus and small white, grey or green flocculi. This fluid flows intermittently and in small amounts, and during this stage it is necessary frequently to clear the tube of flocculi by syringing in a few millilitres of saline. When the tube tip enters the duodenum, the flow becomes more continuous and copious, and the fluid assumes a clear, transparent, greenish-golden appearance without flocculi. The sequence to this point is also that found when intubating patients without obstruction. When obstruction exists, as the tube tip migrates down the small bowel the fluid becomes turbid, brownish and of fæculent smell. (ii) In the remainder of cases the aspirated fluid from the beginning is fæculent and retains this character throughout the migration of the tube. While the tube tip is passing through the duodenum the fluid may temporarily assume a clearer greenish character, which, however, reverts to the fæculent condition as tube migration proceeds. This sequence occurs when regurgitant vomiting has been a feature. These patients are apt to experience copious fæculent vomiting during the swallowing of the tube tip, and they should be assured that such is to be expected.

2. The second observation is a bedside skiagram taken a few hours after the initial passage of the tube. One of six appearances is found (Figure VI). In Figure VIA, the whole of the end of the tube lies in the fundus of the stomach. In Figure VIB, a loop of tube lies in the body of the stomach, but the tip is doubled back towards the fundus. None of the tube extends to the right of the vertebral bodies, and the tip inclines up and to the left. In Figure VIC the tube tip points to the right, but does not extend beyond the vertebral bodies. It is in the pyloric antrum. If the stomach is greatly dilated the tip may project to the right of the spine, but in such a case it inclines downwards and does not exhibit the reversed "S" curve of Figure VID. In Figure VID the tube tip is inclined first upwards across the vertebral bodies and then downwards on the right of these bodies to form a reversed "S". The tip may be associated with a gas bubble. It is lying in the second part of the duodenum. In Figure VIE the lower portion of the tube exhibits a

pronounced reversed "S" curve, the lowest portion obviously occupying the full length of the duodenum. The tip will show a downward inclination if it has negotiated the duodeno-jejunal flexure. In Figure VIF the tube tip is at the same point as in Figure VIE, but the plane of the duodenum is tilted in such a way that an appearance not unlike Figure VIB is seen. In Figure VIB none of the tube extends to the right of the vertebral bodies, and the tube tip points to the left or slightly upwards. In Figure VIF the lower curve of the tube extends to the right of the spine, the tube tip may tend to point downwards, and a gastric gas shadow may be present with the tube tip obviously outside its area.

As subsequent management differs so greatly for the appearances in Figures VIB and VIF, correct radiological interpretation is essential.

If the tube is at the pyloric antrum (Figure VIC), the patient is left undisturbed and another film is taken in six to twenty-four hours.

If the appearance in Figure VIA or Figure VIB is present, the number of centimetres at which the tube lies beyond the external nares is noted in relation to the apparent intragastric length. The tube is withdrawn until it is estimated that the intragastric length is six to eight centimetres.

To assist the tube tip to fall down into the body of the stomach, the patient is kept sitting forward over a cardiac table for half an hour to one hour, after which the tube is again slowly threaded into the 68 centimetre mark. A further film is then taken.

The observations in this paper result from the use of this technique in all cases except Case XI, and with it, passage into the intestine has eventually occurred in 85% of cases. However, three facts have become apparent from repeated observation of the X-ray appearances associated with this method of intubation. (i) If the tube tip occupies the position A (the most common) or B in Figure VI, it is apt, in quite a considerable proportion of cases, to remain there for some days or until the tube is withdrawn. (ii) If the tube tip assumes a position as in Figure VIC, it will, in the majority of cases, pass through the pylorus if given time. (iii) Once in the duodenum, the tube tip will practically always pass without trouble down to the most proximal obstruction.

The tendency for the tube tip to pass to the left and up into the fundus is due to the inclination of the relatively rigid terminal part of the Miller-Abbott tube to pass in the direction of the prolongation of the abdominal œsophageal curve; to the fact that the walls of the body of the stomach normally tend to remain opposed and those of the fundus to remain atonic and distended with an air bubble; and thirdly, possibly, to a shelf formation, below the cardia, of the posterior stomach wall, which is apparently responsible for the "cup and spill" effect occasionally found on fluoroscopic examination of otherwise normal stomachs.

The reluctance of the tube tip to leave this situation is related to (*a*) the physiological behaviour of the stomach wall, and (*b*) the degree of small-bowel distension.

With regard to (*a*), Best and Taylor (1943) make the following observations:

> When the stomach is empty, the cavity of the body and antrum is obliterated by apposition of the walls of these parts. The fundus is dilated with gas. Food, on emerging from the cardia, collects just above the obliterated portion. Apparently simply for its own weight it gradually separates the gastric walls, and passes down into the body. The fundus, and usually the remainder of the stomach above the middle of the body, shows no peristaltic activity. The muscle of this part is the seat of weak tonic contraction which is immediately inhibited by the entrance of food into the stomach. . . . The tonic contraction of the fundic walls does not serve to press the food towards the pylorus. . . . . The stomach above the middle of the body shows no peristaltic activity. Peristalsis, weak at first, commences at this point and becomes more vigorous as it descends.

With regard to (*b*), as small bowel distension develops it exerts an increasing pressure on the stomach wall, which initially is most pronounced on the pyloric antrum and body, squeezing these against the under-surface of the liver (Wild, 1948). This results in their contents being forced up into the fundus. It hinders the regurgitation of intestinal contents from duodenum to fundus, as well as the passage of any form of tube from fundus to pylorus. The greater the distension, the greater these disabilities are likely to be. Consequently, as a post-operative measure it is advisable to anticipate this by instituting intestinal intubation at the first suggestion that an ileus may be developing. This is a reason why, in the early stages of intubation of a tensely distended abdomen, the aspirated fluid is apt to run intermittently and in small quantities. However, once some decompression has been attained, regurgitation from duodenum to stomach usually becomes more free.

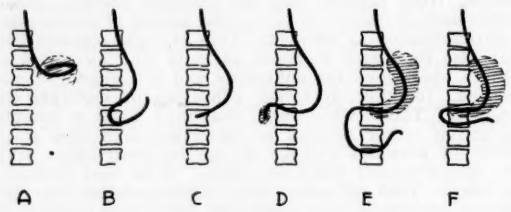


FIGURE VI.

The X-ray appearances that may be found in the early stages of Miller-Abbott intubation. A, tube tip coiled in fundus of stomach. B, tube in body of stomach, but tip doubled back towards fundus. C, tube tip in the desired position—that is, in the pyloric antrum pointing towards the pylorus. D, tube tip in the duodenum. E, tube tip at duodeno-jejunal flexure. F, tube tip at the flexure, but lying in a tilted duodenum, giving an appearance resembling B.

If, therefore, the tube tip assumes a position as in Figure VIA, it appears that the only factor that will swing it down into the body is its own weight; while factors hindering this movement are (*a*) undue rigidity of the terminal few inches of the tube, (*b*) a length of intragastric tube neither short enough to swing down without hindrance by the greater curvature nor long enough to allow it to swing up and over through a circle and so down into the body, and (*c*) apposition of the walls of the body of the stomach.

It would therefore appear, firstly, that the soft, flaccid, heavily-weighted end of the Honor-Smathers tube or of the Cantor tube is preferable to the more rigid terminal sections of the Miller-Abbott and Harris tubes; secondly, that the length of intragastric tube needs to be carefully adjusted according to the appearances in serial skiagrams; and thirdly, that in certain cases it may be desirable to attempt to distend the body of the stomach to allow the tube tip to fall into it.

In consequence, the following technique has recently been given a trial and has given encouraging results (Case XI).

A Miller-Abbott tube is modified to resemble an Honor-Smathers tube in the following manner. The tip of the tube and all lateral bag-filling holes except the most distal one are occluded, and the terminal half of a condom is tied on so that the remaining bag-filling hole communicates with its interior and the major part of the condom hangs from the end of the tube. One or two millilitres of mercury are run into the bag, and with the patient sitting up and inclined forward, the well lubricated bag is passed slowly through the nose into the stomach to the 60 centimetre mark. The tube is strapped to the cheek, but no suction is applied. A skiagram is taken.

If the appearance resembles that in Figure VIA or VIB, an estimation is made from this film of the length of the tube within the stomach. The tube is then withdrawn so that the intragastric length of tube *plus* bag approximates six to eight centimetres (in otherwords, is short enough to allow its end to swing down point first). After thirty minutes, when it is presumed that the end has fallen into the body of the stomach, the tube is passed slowly in to the 68 centimetre position and fixed there, and a further film is taken. This is likely to show an appearance similar

to that in Figure VIc, D, E or F, and if so, continuous suction is commenced. If the tip of the tube is still in the fundus and it appears that the body of the stomach is empty and will not open out to receive it, the tube is partly withdrawn as before, and two tumblers of lukewarm water are given. The tube is again threaded in to the 68 centimetre mark, the tube is strapped to the nose, and a skiagram is taken thirty minutes later.

Wild (1948), using a similar technique, intubated "20 successive cases of intestinal distension in an average time of 57·2 minutes from nose to X-ray evidence of the tube in the duodenum". He withholds suction until the duodenum is intubated and in all cases uses air injection to distend the body of the stomach.

The writer's experience suggests that the major delay in the stomach occurs as described. It appears also that, once the tip of the tube is pointing towards the pylorus and has entered a section of the body of the stomach where it may be grasped by a peristaltic wave, it is merely a matter of time (usually not very long) until the tip passes through the pylorus.

In other words, the function of the surgeon is to provide the optimum conditions to allow the stomach movements to pass the tube tip into the duodenum. It is not the surgeon but the stomach that passes the tube, and this it will do, if permitted, in 85% of cases. Therefore, once an appearance resembling that in Figure VIc is observed, the tube is left undisturbed until a later film shows the tube tip to be in the duodenum.

It has been suggested that the tube tip may be "milked" through the pylorus under X-ray screening control. Sisely reports that attempts to do so precipitated vigorous antiperistaltic rejection by the prepylorus. Even at laparotomy it is often difficult to manipulate the tube through the pylorus.

With the unmodified Miller-Abbott tube technique, it is commonly found that the tube tip enters the duodenum within three to thirty-six hours. It may not do so for some days (four in Case II), and during this period the decision not to operate may be a difficult one to make. If signs suggesting strangulation obstruction are persistently absent, and if the distension is diminishing even though not disappearing, purely gastric suction is persevered with. It alone may prove adequate to allow time for an obstruction from plastic peritonitis to resolve, or the tube tip may eventually negotiate the pylorus.

With this technique, if the patient is allowed sufficient time, it should rarely if ever become necessary to consider the advisability of passing the tube through a gastrostomy or an enterostomy opening.

Mercury is used with an entirely different object from that of Harris and Cantor, who use it in larger quantity as the sole bag content, and to facilitate the passage of the tube both through the pylorus and along the intestine. It is here used solely to encourage the tube tip to fall into the body, and so come within the grasp of gastric peristalsis. A small amount is used, and once the tip is through the pylorus water is injected to distend the bag to a degree sufficient to form a satisfactory bolus.

The phenomena accompanying passage of the bolus and the tube down the small bowel conform to one of three patterns: (i) small-bowel obstruction without a closed loop; (ii) small-bowel obstruction with a closed loop; (iii) large-bowel obstruction with an incompetent ileocaecal valve.

Small-Bowel Obstruction without a Closed Loop.

When the tube tip is demonstrated to be in the duodenum, the balloon is partially filled by injection of water of an amount decided upon by trial before the tube is passed. Over-distension may result in a balloon of such diameter and tenseness as to provide an adequate stimulus to initiate intussusception (Harris, 1945; Smith, 1945). (Case XI.) One aims merely to provide a soft, jelly-like bolus that can be grasped and moved on by peristalsis. A completely empty balloon at times constitutes an adequate stimulus, so that as soon as it negotiates the pylorus, the duodenum starts to pass it on (Cases VII and X). The balloon thus constitutes a bolus which is propelled along the intestine by peristalsis, the tube being threaded through the nose as required. If sufficiently intelligent, the patient has the principle of the tube explained to him and can be relied on to feed the tube in himself. There is a characteristic ease in this feeding-in of the tube which is rapidly appreciated by those performing it, especially if the patient or the nurse has felt the characteristic tug of peristalsis on the balloon. The temptation to thread the tube in too rapidly must be resisted. The rate of progress of the balloon down the small bowel varies considerably. An important cause of delay, readily overlooked, is hypoproteinæmia, and its correction alone may result in rapid acceleration of decompression (Leigh, 1942). Periodic X-ray examinations are desirable for observation of the rate of migration and the point of cessation of migration of the tube tip, and also of the fluctuations in the degree of distension of the various bowel loops.

After the passage of the balloon from a decompressed to a dilated loop of bowel, evacuation of the latter's air and fluid content occurs; but if distension is pronounced, the balloon remains stationary until the bowel recovers its tone and contracts down upon it. When this occurs the balloon stimulates peristalsis and is propelled further. In obstruction occurring in a previously normal bowel (for example, paralytic ileus and post-operative plastic adhesive obstruction), this shrinkage of the bowel diameter (in the absence of vitamin *B* lack and hypoproteinæmia) occurs fairly rapidly. However, if the obstruction is one occurring as a culmination of many previous attacks, the bowel above the obstruction may be permanently dilated (as distinct from "distended"). This dilatation is maximal just above the obstruction and gradually decreases proximally. It appears that in such a case the balloon will be propelled distally to the point where, after evacuation of its contents, the dilated bowel is unable to shrink sufficiently to grasp the balloon. The stimulus to peristalsis disappears and at operation the distended balloon is felt lying free in the dilated lumen, usually some feet proximal to the point of obstruction (Cases II, IV, V and VIII). Although dilated, the bowel is not distended in the acute obstruction sense; but a skiagram may reveal large air-containing loops interpreted as "distended". Such an appearance of "distended coils" *plus* failure of complete migration of the balloon must be interpreted with regard to a past history of obstructive attacks, and such are compatible with completely satisfactory decompression (Cases II and IV).

Sudden increase in the flow of aspirated fluid and gas commonly follows an attack of colic and borborygmi, which in turn sometimes results from movement of the patient in bed. Active movement of the patient and gentle abdominal massage by the patient or nurses facilitate the unkinking of bowel loops rendered possible by decompression proceeding satisfactorily.

The changes in the aspirated material are best observed in the glass Y-tube, and this is often watched with interest by the patient. He is frequently asked to watch and report certain changes, such as sudden cessation of flow at a stage when it should not occur. The enlistment of his services is often of help and certainly improves his cooperation. Where intubation proceeds satisfactorily, it is usually found at the start that a copious flow of fluid in gushes alternates with gas in the rough proportion of one to three volumes. The gas may at times come away in rapid bursts of large volume. As decompression proceeds, the proportion of gas steadily diminishes until it becomes minimal when decompression has been completed. It is this gas that makes the top suction bottle empty so rapidly in the early stages, and makes one of large capacity so desirable; and it is the disappearance of this gas that makes infrequent filling of the top bottle necessary when decompression has been attained.

During the progress of decompression the aspirated fluid is turbid brown and of fæculent smell. When all this stagnant fluid has been aspirated the fluid becomes thin, transparent and golden, and represents normal intestinal secretion. This appearance persists so long as the fluid is aspirated as soon as it is formed. If cessation of aspiration from tube block occurs, the fluid accumulates in the bowel and gradually assumes a fæculent character, the amount and degree of change being proportional to the

period of interruption of aspiration. The fluid aspirated after restoration of suction is therefore at first fæculent, and this should not necessarily be regarded as evidence of clinical deterioration.

If multiple mechanical blocks are present from kinking, unfolding of the most proximal kink may be so long delayed that complete emptying of the bowel above is attained with change of the fluid to normal intestinal secretion. The kink may then unfold, and this will manifest itself by a sudden increase in the rate of aspiration with a sudden change to fæculant characteristics and possibly the aspiration of gas. This irregular alternation may occur several times until the tube tip reaches the most distal block.

Completion of Decompression.

There are two distinct phases in suction decompression, (*a*) that of establishment of decompression, and (*b*) that of maintenance of such decompression until the obstruction is relieved spontaneously or by operation. The completion of the first phase is indicated by the disappearance of clinical and radiological evidence of distension, by cessation of pain, and by a change of the aspirated fluid to a clear light golden-brown fluid, which comes away freely and steadily in good quantity. This stage is often confused with that of relief of obstruction, with consequent premature termination of suction and subsequent reappearance of the features of obstruction.

Resolution of the Obstruction.

Relief of the obstruction is suggested by a material fall in the daily amount of fluid aspirated, and by the passage of fæces and flatus.

It soon became apparent, in waiting for obstruction to resolve while intubation decompression was maintaned, that normal patency and movement of the bowel are present only intermittently for a varying period after their first appearance. In other words, the passage of fæces and flatus cannot be regarded as evidence of complete and lasting resolution. Early in our experience we removed the Miller-Abbott tube as soon as this occurred, only to find a recurrence of colic, abdominal distension and sometimes vomiting. These phenomena are well seen in Cases II, III and VII.

The following procedure for terminating intubation is therefore recommended. If fæces and flatus are passed and the amount of fluid aspirated materially decreases, the Miller-Abbott tube is clipped off, oral fluid administration is encouraged, and the patient is observed for at least twenty-four hours. If, during this period, there is no recurrence of pain, abdominal distension, nausea or vomiting, and if operation is not contemplated, the tube is removed. If any doubt exists the tube is left in position for an extra day.

It is to be emphasized that even repeated spontaneous passage of fæces and flatus does not necessarily indicate permanent resolution of the obstruction.

If the clipping off is not tolerated, suction is recommenced and continued until decompression is again attained, at which time the decision will have to be made whether a further delay for possible permanent resolution is justified.

The duration of the two phases of suction decompression varies considerably. Resolution of obstruction may occur before complete decompression has been attained.

Small-Bowel Obstruction with a Closed Loop.

The presence of a closed-loop small-bowel obstruction is usually not apparent at the commencement of intubation. As the small bowel above the proximal block becomes decompressed, the collapse of this bowel allows the characteristic X-ray appearance and clinical features previously described to reveal themselves. Treatment is then modified along the lines that have already been indicated.

Large-Bowel Obstruction with an Incompetent Ileo-Caecal Valve.

If, after satisfactory migration of the tube tip to the ileum, and initial satisfactory decompression, the fluid commences to run intermittently and with difficulty, takes on the colour, turbidity and smell of fæculent fluid but assumes the consistency of thin cream, then the fluid is to be described as "fæcal" as distinct from the watery "fæculent" of small-bowel obstruction, and is to be regarded as truly fæcal content that has been regurgitated through an incompetent ileo-caecal valve and as the result of colonic obstruction (Case IX).

In no case of intubation has it been found necessary to use gastric suction in addition to intestinal suction, or to withdraw a satisfactorily migrated Miller-Abbott tube to a higher level to decompress more proximal intestine.

The procedure of intubation suction decompression requires frequent observation, at least throughout the phase of attainment of decompression, by the surgeon himself. It should not be left to the unsupervised management of a house surgeon.

The Maintenance of Nutrition.

A major danger in intubation treatment is that of mismanagement of the problems of nutrition, the term being used in the broad sense to include water, salt, protein, carbohydrate, fat and vitamin requirements. Such mismanagement is inevitable and may be lethal if suction is applied, even though successfully from the standpoint of the obstruction, without careful replacement of deficient elements present at the commencement of treatment and carefully calculated maintenance of nutrition through the period of suction. An accurate record of all intake and output is kept on a special fluid balance chart (Gale, 1946). So long as the tip of the tube lies proximal to the duodeno-jejunal flexure, nourishment must be given parenterally.

At the commencement of treatment, an attempt is made to estimate roughly from the patient, his relatives and his family doctor the amount of fluid that has been lost by vomiting. The salt and water depletion so determined is corrected by the intravenous administration of one pint alternately of normal saline and of 5% dextrose solution in water, the rate of administration being regulated according to the severity of the depletion. The water, salt and glucose requirements are reassessed the next morning. Though empirical, this procedure proves satisfactory in practice.

It is necessary to emphasize that Coller and Maddock's "clinical rule" is not recommended. This is as follows:

> For each 100 milligrams that the plasma chloride level needs to be raised to reach the normal of 560 milligrams per cent., the patient should be given ·5 gram of sodium chloride per kilogram of body weight.

Coller and Maddock advocated this rule in both 1938 and 1940. In 1944, however, Coller and others (1944) retracted the rule with these words.

> These cases have convinced us that the use of the "clinical rule" is dangerous . . . and that it should not be employed. It is recommended that the correction of fluid deficiency states be made upon the basis of the physiologic response to test doses of the appropriate salt solution rather than upon the basis of the plasma chloride, CO_2 combining power, N.P.N., plasma protein or hæmoglobin levels.

The writer agrees with this view. Unfortunately, a considerable number of writers (Edwards, 1948; Dennis, 1949; Mason and Zintel, 1946) continue to quote and advocate this rule and do not appear to be aware of Coller's later work and retraction.

Also at the commencement of treatment, the degree of starvation, in particular in respect to protein and vitamins, is assessed. This may suggest the advisability of serum or blood transfusion. Vitamins *B* and *C*, because of their influence on gastro-intestinal mobility and healing respectively, are given by injection in large doses.

The calculation of the water, salt and glucose requirements for each subsequent day has to provide for loss of water during the next twenty-four hours by vaporization from skin and lungs and by way of the urine. This is

provided for in the average case by 60 ounces of a 5% solution of dextrose in water. In addition, the loss of fluid by vomiting and aspiration in the preceding twenty-four hours is made up in the next twenty-four hours by the administration of normal saline in equal amount. If the quantity so lost is large, it is better not to attempt to replace it all, as the fluid lost is usually not quite isotonic.

In general it is better to give too little rather than too much. The best guide to the state of hydration is the daily urinary output. If it exceeds 35 ounces of urine with a specific gravity of 1015 or more in the absence of glycosuria, hydration is probably being adequately maintained. Fantus's test for urinary chlorides is a useful confirmatory guide to the chloride intake.

The inevitable protein starvation is provided for by the administration of serum every third day or by a suitable amino-acid preparation. For a more detailed discussion the reader is referred to another paper by the writer (Gale, 1946).

Once the intestinal tube has passed the duodeno-jejunal flexure, parenteral therapy can frequently be discontinued and nutrition maintained by oral feeding (Penberthy *et alii*, 1940). This may be commenced even though decompression is not complete. The administration of a non-residue liquid diet is commenced cautiously, the patient's tolerance being the guide to its continuance. During oral feeding the suction apparatus requires careful watching for blockage from flocculant material resulting from the theoretically non-residue diet. Oral feeding is used even when operation is regarded as inevitable (Cases II, III, VIII and IX).

A non-residue diet rich in protein, carbohydrate and vitamins, (especially the *B* complex and vitamin *C*) is used. The lower the tube tip is in the small bowel, the more varied the food allowed. With the tip high in the jejunum, protein and salt absorption is likely to be inadequate (Wilensky, 1945). (Case VII.)

It is preferable to continue intravenous fluid administration during the first day or two of oral feeding, in case such feeding should prove premature, and because even if tolerated it will be insufficient at first to provide for the fluid requirements.

The patient is allowed salt according to taste, it being believed that this will result in satisfactory adjustment of any abnormal variation in the total salt content of the patient at the time of discontinuance of parenteral therapy. (Case III.)

REPORTS OF CASES.

The following case reports have been selected to illustrate the value, the pitfalls and various points in the management of intubation in intestinal obstruction. Two cases ended fatally.

CASE II.—D.S., aged seventeen years, was admitted to hospital on December 29, 1945, with abdominal colic and vomiting. After appendicectomy in 1936 he had suffered from recurrent attacks at three-monthly intervals of constipation, severe colic and recurrent dry retching. The present attack had commenced three days previously with pain and flatulence and later vomiting (which became persistent and fæculent), and abdominal distension and constipation not relieved by enemata.

Examination of the patient revealed considerable dehydration, a subnormal temperature and a pulse rate of 110 per minute. The abdomen was much distended with an obvious ladder pattern. Tenderness was generalized, but most pronounced in the epigastrium, where there was slight rigidity. No mass was palpable. A Miller-Abbott tube was passed and intravenous therapy was commenced. Two hours later, after a drink of water, 10 ounces of fæculent fluid were vomited.

During the next eleven hours no fluid was aspirated. Inspection of the glass Y-tube showed that a continuous stream of air bubbles was being sucked over. On listening near the patient's nose, a continuous suction sound could be heard. The patient complained of discomfort at the back of the nose, and inspection revealed the coiled up Miller-Abbott tube impacted in the naso-pharynx. This had apparently followed the vomiting eleven hours earlier. The tube was withdrawn and reinserted, this procedure being accompanied by the vomiting of 15 ounces of fluid. No further vomiting occurred.

The following day there was no pain and distension was less. The next day he was comfortable, but distension was greater.

On the following day, January 2, 1946, an X-ray film showed the tube tip in the stomach with small-bowel distension persisting in one area. At 7 p.m. the advisability of operation was considered, as the colicky pain had recurred; the temperature, which had been gradually rising, was 102·4° F. and the pulse rate 100 per minute; an X-ray examination revealed persistent small-bowel distension with the tube tip still in the stomach. However, the suction apparatus was working well and had removed 330 ounces of fluid. No mass was palpable. The rise in temperature was regarded as being due to phlebitis from the intravenous therapy. The fact that on two occasions on this day and on two occasions on the previous day the pulse rate had fallen to 84 per minute was regarded as being against the probability of strangulation. (Subsequent cases proved this to be fallacious reasoning.) Operation was not performed. The temperature fell by lysis, and from January 5 to the time of operation on January 8 the pulse rate never exceeded 84 per minute.

On January 3 the fluid removed by suction was biliary in the early morning, but later changed to more offensive fæculent fluid. The patient became more comfortable and the distension less. At 5.30 p.m. X-ray examination revealed the tube tip in the third part of the duodenum. The Miller-Abbott tube thereafter passed steadily down the intestine. The aspirated fluid became much thinner and clearer, and the distension decreased.

On January 4 the bowels were opened twice with normal stools, and the administration of a non-residue diet rich in protein and vitamins was commenced.

On January 6 the suction stopped flowing for ten hours, with recurrence of distension and pain. Suction was reestablished, 98 ounces of brown fæculent fluid being aspirated in the next three hours with rapid relief of the symptoms.

On January 8 elective operation was performed with the tube *in situ* and the suction apparatus working. The bowel was found to be flaccid and collapsed (Figure VII).

The fundus of the Meckel's diverticulum was attached by a solid fibrous cord to the deep surface of the umbilicus, the cord being twisted in a clockwise direction. The mesentery of the diverticulum was attached by a tight, attenuated, fibrous extremity to the left side of the ileal mesentery adjacent to the bowel. The structures extending from this point to the umbilicus were taut. Where it crossed the ileum, the attenuated end of the diverticulum mesentery formed a tight fibrous band incorporated in the ileal wall. At this point there was a narrow fibrous stricture involving the whole ileal circumference. The bowel proximal to this point was flaccid, thickened from hypertrophy, and three inches in diameter.

The hypertrophy and dilatation gradually diminished proximally, but were still pronounced where the distended balloon of the Miller-Abbott tube was felt through the bowel wall four feet above. The bowel distal to the stricture was normal, the diverticulum neck being three inches from the obstructing band. This length of bowel and the free margin of the diverticulum mesentery formed a hole through which a twelve-inch loop of flaccid small bowel projected. This loop manifested no evidence of recent obstruction, but there were numerous old filmy adhesions between these bowel loops and between the folds of its mesentery. Such appearance was not present in the remaining small bowel.

As the stricture under the band involved the whole thickness and circumference of the bowel, resection of the bowel and the diverticulum in one piece followed by side-to-side anastomosis was performed. The abdomen was closed after the end of the Miller-Abbott tube had been threaded a few inches past the anastomosis. The procedures were performed with ease in a non-distended abdomen and on bowel manifesting no suggestion of obstructive circulatory embarrassment.

At operation, the balloon of the Miller-Abbott tube was found satisfactorily distended and lying free in bowel of a greater diameter. Apparently the balloon acted as a bolus and a stimulant to peristalsis so long as its external diameter exceeded that to which the bowel was capable of contracting after the latter had been decompressed. As soon as the balloon entered bowel affected by chronic

dilatation (as distinct from distension) of such degree as to make it incapable of contracting down on the balloon, the balloon ceased to constitute a bolus and therefore failed to migrate further. However, such conditions proved compatible with the attainment and maintenance of satisfactory decompression.

After operation, profuse drainage continued through the Miller-Abbott tube (to midnight, 79 ounces of blood-stained fæculent fluid).

On January 9 the patient was comfortable, the abdomen was soft and flat. Suction removed 82 ounces of blood-stained fæculent fluid. On January 10 oral feeding with fluids and food was followed by spasms of pain. Suction removed 129 ounces of fæculent fluid. On January 11 the patient was feeling well, but having frequent attacks of slight colicky pain. Slight distension was present. A good constipated motion followed a glycerin enema. Suction removed 75 ounces of brown fæculent fluid, which was slightly turbid.

On January 12 some spasms of pain were felt at times, but the aspirated fluid was clearer. The diet was increased. Suction removed 60 ounces of greenish-brown fæculent fluid. On January 13 the patient was not feeling so well, and complaining of frequent severe colicky pains. He was nauseated after a meal. Suction removed 50 ounces of greenish-brown fæculent fluid. On January 14 he was more comfortable. He was given an emema in the morning, and a light-brown semi-solid stool was passed. During the day he had four spontaneous bowel actions. The Miller-Abbott tube was clipped off. Suction removed 20 ounces of light-brown fluid.

On January 15 the bowel was open with the passage of flatus. The Miller-Abbott tube was removed. On January 16 the administration of a diet rich in protein was commenced. On January 17 the patient had flatulence and pain at times. On January 19 a rectal tube was inserted and flatus was passed. On January 20 some severe spasms of pain and a little distension were present. An enema was given, with a fæcal result and relief of symptoms. There was no further trouble.

In October, 1947, the patient reported that he had been well and symptomless.

The following points are of interest:

1. The impaction of the Miller-Abbott tube in the naso-pharynx after a vomiting attack.

2. The four-day delay in the passage of the Miller-Abbott tube through the pylorus, that period being associated with aspiration of 474 ounces of fluid varying irregularly from fæculent to clear green and then to fæculent, and with only partial relief of symptoms.

3. The change in the aspirated fluid back to fæculent characteristics, and the recurrence of pain and distension following ten hours' interruption of suction; this also indicated that, in spite of two bowel actions two days previously, the abnormality causing the obstruction had not resolved.

4. The radiological persistence of small-bowel "distension", due not to obstructive distension but to chronic dilatation accompanying chronic obstruction.

5. The elective operation performed because of the obviously chronic nature of the obstruction, the technical ease of the operation and the interesting pathological findings.

6. The value of the post-operative suture line protection by the indwelling Miller-Abbott tube, the desirability of this being indicated by the post-operative undue persistence of a fæculent character of the aspirated fluid and the unusually large amount of this fluid, and by the recurrent attacks of colic and distension.

CASE III.—R.R., aged 50 years, was admitted to hospital on September 5, 1944, with the diagnosis of "abdominal pain—query hysteria". After the removal of an acutely inflamed appendix two years previously, she had suffered from recurrent attacks of abdominal pain and borborygmi without vomiting. The present attack had commenced eight hours previously with severe cramping pains in the right side, which passed through to the back. There was nausea, but no vomiting. The abdomen was neither rigid nor distended, but vague tenderness was detected under the right costal margin. She was regarded, as on previous occasions, as suffering from a neurosis.

During the next five days, attacks of pain recurred. The patient vomited for the first time on September 9 amounts of two, six and eight ounces of yellow fluid. On September 10 she vomited fæculent fluid. Her bowels had opened twice on the day of her admission to hospital, but not since. Her pulse rate had gradually but steadily increased from 68 per minute on her admission to 120 on this morning. After the fæculent vomiting, repeated bowel washouts (resulting in a clear fluid return) were given, intermittent (four-hourly) suction through an indwelling stomach tube was instituted, and continuous intravenous fluid administration was commenced.

Twenty-four hours later (September 11), as very little fluid had been aspirated and no vomiting had occurred, the stomach tube was removed. During the next three days the patient frequently vomited a few ounces at a time of "brown fluid". On the morning of September 13 the abdomen was described as "soft and not distended". At 7 p.m. she was transferred to the writer's care.

The abdomen was distended. An X-ray film showed distended loops of small bowel. Rectal examination revealed tenderness in the right side of the pelvis with a suggestion of a mass. Continuous intravenous therapy was recommended and a Miller-Abbott tube was inserted, 56 ounces of fluid being aspirated within a few hours. She did not vomit again.

During the next five days large quantities of fluid were aspirated and the tube was passed into the ileum. On September 18, although the abdomen was flat and only slightly tender, she still complained of occasional stabs of pain. On September 19 an X-ray film showed the tip of the tube at the right side of the pelvis and an absence of air-containing bowel loops. The administration of a non-residue diet was commenced.

On September 20 she passed flatus during the morning. At 3 p.m., 5 p.m., 7 p.m. and 10.30 p.m. she passed small constipated stools and flatus. As fluids were being taken orally, intravenous therapy was discontinued.

On September 21 an X-ray film showed the tube tip in the same position. A dark-brown fluid stool was passed. During the day 86 ounces of fluid were aspirated.

On September 22 some slight abdominal pain was complained of. A fluorscopic examination with barium injection through the Miller-Abbott tube gave the appearance shown in Figures VII and VIII. At 5.30 p.m. the suction apparatus was clipped off. At 9.30 p.m. she felt "full". At 10 p.m. she complained of nausea and discomfort, so the suction was resumed, this being followed by rapid aspiration of 44 ounces of fluid.

On September 23 at 1 a.m. the suction apparatus was clipped off. At 11.30 a.m. an enema gave a very good result of fæces and flatus, and at 2 p.m. a brown fluid stool was passed. At 9 p.m. suction had to be restarted owing to some hours of discomfort and "wind in the stomach". However, she was feeling well and had developed an unusual desire for table salt, which she was taking in pinches, in water and in orange juice.

It was concluded that a non-strangulating small bowel obstruction due to an adhesive band was present, which was adequately decompressed and had partially resolved, but which was showing no evidence of resolving to a degree sufficient to warrant further conservative treatment. Suction decompression and oral feeding with a non-residue diet were therefore continued until elective operation was performed on September 26.

At operation, all bowel loops were found collapsed and flaccid, so that easy exposure was obtained of the condition shown in Figure IX.

A tense conical band, with the base at the caecum at the junction of the longitudinal muscle bands, stretched transversely anterior to four pieces of small bowel, to be attached to the left side of the ileal mesentery one inch from the latter's attachment to its bowel. All four pieces of bowel immediately beneath the band were affected by localized fibrosis and some contracture, and were partially adherent to one other. The most proximal constriction was four feet from the ileo-cæcal valve, the bowel immediately above being chronically dilated and hypertrophied, and that distal thereto being slightly hypertrophied. The tip of the Miller-Abbott tube was nine inches above this constriction. These appearances were well suggested in the skiagram (Figure VIII). The apex of the conical band was divided, after which it disappeared by retraction on to the surface of the caecum (Figure IX). The localized adhesions on the loops distal to the most proximal obstruction were separated and the tip of the Miller-Abbott tube was threaded past the proximal obstruction.

The sequence of events thus appeared to have been the excision of an acutely inflamed appendix followed by adhesion of its divided base to the ileal mesentery, gradual elongation of the adhesion, recurrent spontaneously resolving obstruction at all four points of bowel crossed by the adhesion with resultant inflammatory reactions at the point of constriction and hypertrophy of the involved loops, and finally, an obstruction failing to resolve and requiring surgical intervention.

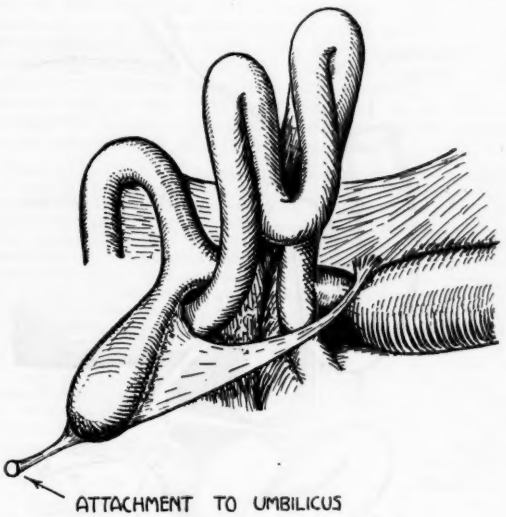


FIGURE VII.

Case II (D.S.), showing the attachments of the Meckel's diverticulum and the resulting obstructions.

The post-operative convalescence was uneventful. On the fourth day the bowels were open normally, after which the suction apparatus was clipped off. The Miller-Abbott tube was removed next morning.

Several months later the patient again experienced partial obstruction, which resolved without operation.

The following points are of interest:

1. The striking diagnostic value of the barium injection shown in this case is unfortunately not a common experience.

2. The criteria indicating the various stages in suction decompression are well shown. Satisfactory decompression was readily obtained, but resolution of the obstruction never became more than partial and remained insufficient, without the aid of the tube suction, to decompress the bowel above. That partial resolution of the obstruction occurred is indicated by the barium shown in the non-distended bowel loops beyond the obstruction and by the frequent apparently satisfactory bowel actions. That the resolution was inadequate was shown by the fact that the daily amounts of fluid aspirated remained high (68, 86 and 44 ounces successively) after these bowel actions had occurred. These are to be compared with the amounts of three, four, 16 and 20 ounces after the band had been divided.

That the relief of obstruction was inadequate is also indicated by the recurrence of discomfort, "wind" and nausea each time the Miller-Abbott tube was clipped off, by the large amount of fluid aspirated in a short period, and by the relief of symptoms following resumption of suction. The necessity for leaving the tube in position until one is absolutely certain that it is no longer necessary is thus indicated.

3. The elective nature of the operation is evident. It was performed on a fully hydrated and nourished patient, on a lax abdomen containing retractible healthy bowel, and with a means of post-operative decompression (the Miller-Abbott tube) in position and capable of giving protection in any major procedure that might have been found necessary.

4. In view of Marriott's (1947) statement that no specific sensory warning of salt depletion occurs in man, the patient's behaviour on September 23 is of interest. On September 20 intravenous therapy was discontinued owing to the legs becoming painful and œdematous. The

FIGURE VIII.

Case III (R.R.). Skiagram obtained after injection of barium through the Miller-Abbott tube.

administration was not transferred to another limb, as the occurrence of several bowel actions and X-ray proof of satisfactory migration of the Miller-Abbott tube suggested that relief of the obstruction was imminent, with resulting opportunity to give adequate fluid orally. Such hope was not realized, and during the next four days ten pints of fluid were aspirated. This was replaced by the oral administration of fluids and a standard non-residue diet. On September 23, water depletion having been corrected by voluntary oral intake, a sudden abnormal appetite for salt became manifest and lasted two days. Salt was ingested not only as a condiment, but voluntarily as solid salt and in orange juice and in water. Such appetite (which the patient admitted was bizarre) was encouraged, as it appeared a natural mechanism to relieve salt depletion resulting from the unprovided-for loss in alimentary suction.

CASE IV.—A.G., aged forty-nine years, a male patient, had had recurrent attacks of colic and abdominal distension since an operation for adhesive obstruction a few years before. The attack consisted of ten hours of pain and fæculent vomiting. The pain, at first colicky, had become continuous, and was present in the left side of the abdomen, where some rigidity and tenderness were detected. Abdominal distension was generalized and pronounced. X-ray examination revealed irregular small bowel distension. A Miller-Abbott tube was passed, and X-ray examination twelve hours later showed it to be in the duodenum. It rapidly migrated down the bowel. X-ray examination on each of the next two days showed the tube to remain confined to the upper and right quadrant of the abdomen. Distension of the coils of bowel in the left iliac fossa was less but still present, as was some tenderness. These features suggesting a persistent closed-loop obstruction, operation was performed under "Pentothal" anæsthesia. A large band of thickened omentum was found forming a snare which loosely

encircled a long loop of small bowel showing evidence of old peritoneal damage. The proximal part of the bowel was chronically dilated and hypertrophied, but all bowel was flaccid and readily held away from the loose obstructing band which was divided. The abdomen was closed, and two days later the Miller-Abbott tube was removed.

CASE V.—Miss A.W., aged twenty-four years, was admitted to hospital on January 29, 1946, with abdominal pain, vomiting and constipation. Nine months previously elective

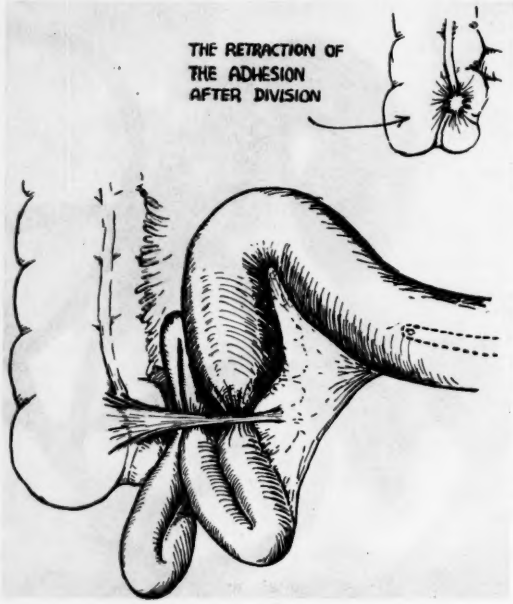


FIGURE IX.
Case III (R.R.), showing operative findings.

right oophorectomy and appendicectomy had been performed, the operation being followed by abdominal pain, high temperature and rapid pulse rate which subsided after some days. Examination of the patient on January 29 revealed a uterus enlarged to the umbilicus, with some soft, tender, resonant distension above and to the right of the fundus, but no rigidity. The temperature was 97·6° F. and the pulse rate 100 per minute.

On January 30 recurrent abdominal colic and repeated vomiting were present. The temperature was 99·4° F. and the pulse rate 120 per minute. An X-ray film was reported on as follows: "Five months pregnancy. A few distended gas-filled loops to right of umbilicus. No gas in colon."

A stomach tube was passed, and the stomach was intermittently aspirated. The tube was vomited five hours later.

On January 31 the vomiting and pain persisted without relief.

On the morning of February 1 the Rehfuss tube was reinserted and intermittent aspiration was commenced. The pain and vomiting persisted and the distension increased.

At 6 p.m. on February 2 the patient was transferred to the writer's care, and the Rehfuss tube was replaced by a Miller-Abbott tube to which continuous suction was applied. She did not vomit again. During this period of eighteen hours, during which intermittent aspiration through the Rehfuss tube was used, the patient's condition deteriorated, and although 69 ounces were aspirated, 26 were vomited.

On the following day (February 3) she felt better, but the pulse rate remained rapid and spasms of pain recurred. At 12 noon an X-ray film showed the tube tip to be doubled back to the gastric fundus. At 8 p.m. (twenty-six hours after intubation) an X-ray film showed the tube to be in the second part of the duodenum. The amount of fluid aspirated for the day was 126 ounces.

On February 4 she was complaining of persistent epigastric pain and soreness, as well as occasional colicky spasms. Examination revealed that, although the abdomen was generally tender, there was a more tender resonant mass to the right of the umbilicus. An X-ray film showed the Miller-Abbott tube to be well down the small bowel and the local area of distension to the right of the umbilicus to be more pronounced. Although 280 ounces of fluid had been aspirated since Miller-Abbott intubation, her condition had deteriorated. There had been a steady upward trend in both temperature and pulse rate. A persisting closed-loop obstruction was diagnosed and laparotomy was performed.

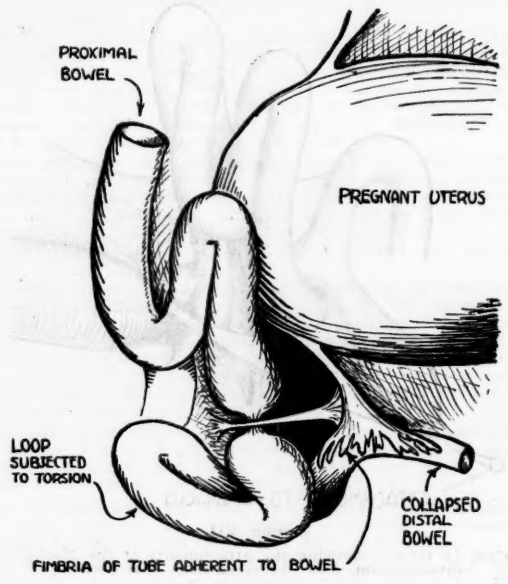


FIGURE X.
Case V (A.W.), showing double small-bowel obstruction from adhesive bands.

The uterus was enlarged to the size of a six months' pregnancy. The fimbria of the right Fallopian tube embraced and was adherent to the small bowel (Figure X). A narrow conical avascular adhesion stretched from the outer upper border of the tube to the right across a loop of small bowel, to be attached to the later's mesentery near its attachment to the bowel. This adhesion was very tight, causing obstruction of the subjacent bowel. The bowel for some feet proximal to this point was dilated and reddened, but not distended, the end of the Miller-Abbott tube being felt two feet above the block. The loop of bowel between this block and the fimbrial adhesions was distended, tense, congested, reddish-purple, and twisted around an axis formed by the adjacent conical adhesion mesenteric attachment and the fimbrial adhesion. The bowel distal to the fimbria was collapsed and of normal diameter and appearance. The conical adhesion was divided at its mesenteric attachment, the volvulus then resolving. The Miller-Abbott tube was threaded down to the fimbrial attachment and the abdomen closed.

The sequence of events appeared to be the development of adhesions following the oophorectomy of May, 1945, and in the few months prior to the present admission to hospital, a gradual stretching and increase in the tension of the conical adhesion, the Fallopian tube, the fimbria and the broad ligament by the enlarging uterus, until a non-strangulating obstruction by external pressure by the conical adhesion and a strangulating small-bowed volvulus were produced. The Miller-Abbott tube satisfactorily decompressed the former and partially decompressed the latter, by allowing increased room (and mobility) for the twisted loop by virtue of the gas and fluid aspirated from the bowel above the first obstruction.

On the patient's return to the ward continuous suction was reapplied and 36 ounces of fluid were aspirated to midnight.

The following day (February 5) her condition was improved, 97 ounces being aspirated. She thereafter rapidly became well, the elevated temperature and pulse rate sub-

siding, and the daily amounts of aspirated fluid were 54 and 34 ounces.

On February 7 the Miller-Abbott tube was clipped off for increasing periods without discomfort, and flatus was passed. On February 8 the bowels opened spontaneously, after which the Miller-Abbott tube was removed. Convalescence thereafter was uneventful.

The enlargement of the uterus precipitating complete obstruction is of interest. The value of the pre-operative decompression of the bowel above the band obstruction and of the correction of the biochemical deficiencies was most apparent, as was the post-operative insurance against ileus provided by the indwelling Miller-Abbott tube. The contrast between the inefficient intermittent gastric suction and the continuous intestinal suction is apparent. The way in which the coincident existence of a closed-loop obstruction became apparent as decompression proceeded is characteristic of this type of case.

CASE VI.—Mrs C.H., aged thirty-seven years, was admitted to hospital on August 28, 1947, at 6 a.m., with a history of having undergone appendicectomy and oophorectomy three years previously and a confinement four months previously. Two months before her admission to hospital she had gradually begun to feel unwell with occasional vague lower abdominal pains and shivers, sweats, and severe headaches. An attack of abdominal colic similar to her previous pains commenced six hours prior to her admission to hospital, and she had vomited twice since. Her bowels had been open twenty-four hours previously. On examination of the patient, the temperature was 97° F. and the pulse rate 72 per minute. Some lower abdominal tenderness and slight rigidity were present. Some borborygmi were heard and were associated with pain. There was no abdominal distension and no visible peristalsis. The leucocytes numbered 14,800 per cubic millimetre. An enema resulted in the passage of fluid flecked with fæces and some flatus. Except for some pain in the late afternoon she had a fairly comfortable day with no vomiting, the four-hourly temperatures being 97°, 99°, 97·4° and 99° F., and the pulse rates being 72, 84, 96 and 76 per minute.

That night she slept well, until she woke at 3 a.m. with colic. She vomited a few ounces of green biliary fluid on each of four occasions during the day, the four-hourly temperature being 98·2°, 98·0°, 97·8° and 98·0° F. and the pulse rate being 84, 72, 76 and 72 per minute. At 6 p.m. on this day (August 29) a Miller-Abbott tube was passed; the temperature was 98.0° F. and the pulse rate 72 per minute.

That night she slept restlessly. She was examined by the house surgeon at 11.50 p.m., when she appeared to be no worse. The suction apparatus was working well and aspirating dark green fluid.

At 3.30 a.m. on August 30 she was more restless, and her extremities were found to be cold and cyanosed and the distension was more obvious.

At 5 a.m. she was examined by the house surgeon. She informed him that at 3 a.m. the colicky pain had changed to a constant pain which was not relieved by morphine. He found that the aspirated fluid was now fæculent, her pulse was barely perceptible, the heart rate being 150 per minute by stethoscope. Her extremities and face were cold and cyanosed; the abdomen was silent, only slightly more distended, generally tender, and firm but not board-like.

At 7 a.m. a small lower mid-line incision was made under local anæsthesia; a small amount of very slightly blood-stained clear fluid, but no gas or other evidence of perforation or peritonitis was found. A drain tube was inserted and the abdomen was closed. In spite of the intravenous administration of serum, the pulse remained imperceptible and the heart rate rapid. The cyanosis and coldness of the skin increased, and the patient gradually became unconscious and died at 1 p.m.

Post-mortem examination revealed a band of omentum adherent to the appendicectomy scar and forming a noose strangulating a loop of small bowel. There were no other obstructed loops. The strangulated loop was four inches long by one and a half inches in diameter. Its surface was smooth, shining and purple. It possessed no adhesions and its surface showed no suggestion of fibrin.

This loop with some adjacent bowel was excised. Careful search of both the serosa and the mucosa revealed no gangrene and no perforation, either patent or plugged. There was no evidence of material extravasation of blood or fluid either into this loop or into any other abdominal viscus. The pelvis was apparently normal. When the abdomen was opened, a small amount of odourless, slightly blood-stained clear fluid was found. No gas excaped. Careful search of the whole peritoneal cavity revealed no pus, no fibrin and no recent adhesions.

A persistently normal pulse rate and temperature may accompany a strangulating obstruction to within a few hours of the onset of irreversible peripheral circulatory failure.

The erroneous diagnosis of partial obstruction of inflammatory origin was made on the long duration and other features of the history.

The cause of the circulatory failure is not clear. The post-mortem findings apparently exclude material loss of serum or blood into the peritoneal cavity, and, from its appearance and size, into the strangulated loop. There was no evidence of bacterial inflammation. If a toxic substance is to be postulated, such a substance was not suddenly released in quantity into the circulation by release of the strangulating agent, because that agent was never released in this case. Was it due to the cumulative action of absorption over several days? If so, the persistence of a normal pulse rate almost to the onset of intense circulatory failure is difficult to explain. How much of such a toxic substance comes from the strangulated loop (in this case so small and so apparently relatively harmless), and how much from the distended bowel above? Knight (1937) suggests that distension of bowel not subject to extramural strangulation results in the appearance of depressor substances in the peritoneal fluid. However, in this case distension was not clinically pronounced until shortly before the onset of the circulatory failure.

CASE VII.—Mrs. E.W., aged forty years, in July, 1947, suffered from pelvic peritonitis in which one foot of ileum was stuck by fibrinous adhesions to Douglas's pouch. The adhesions were separated, some oozing occurring from the separated surfaces. For the following fourteen days suprapubic drainage of pus was intermittent, and was associated with periods of pain and increased temperature and pulse rate. During the next three months she was well. She was readmitted to hospital with a six-day history of obstruction which responded to starvation and enemata. Laparotomy one week later disclosed a fibroid condition of the uterus, chronic salpingitis and extensive small-bowel adhesions. The uterus and Fallopian tubes were removed. Only those adhesions regarded as potentially obstructive were divided. During the next three months she experienced recurrent attacks of pain, vomiting and constipation, a further admission to hospital resulting in resolution without operation. She was again admitted to hospital with similar obstructive symptoms on December 12, 1947. An X-ray examination revealed a distended colon without small-bowel obstruction. Operation disclosed an obstructed but operable carcinoma of the descending colon. The small bowel was not distended. Caecostomy was performed. After a few days of comfort, she began to experience recurrent attacks of colicky pain, borborygmi and abdominal distension, all of which were localized to a small area to the left of the umbilicus. On the sixth post-operative day she vomited for the first time. X-ray examination disclosed moderate small-bowel distension in the left upper quadrant of the abdomen; *valvulæ conniventes* were seen in the coils. From the onset of the pain the caecostomy drainage had become much less.

A Miller-Abbott tube was now passed (December 23), oral feeding was discontinued, and intravenous nourishment was initiated with 25 ounces of blood. At 5 p.m. the following evening (December 24) it was found that nothing had been aspirated since the tube had been passed, and that recurrent vomiting was taking place. Inspection revealed two tubes bridging the pharynx, the Miller-Abbott balloon being impacted in the naso-pharynx. It was withdrawn through the mouth, cleaned and passed without difficulty into the stomach. No further vomiting occurred for one week.

At 9 a.m. on December 25 it was reported that the fluid had changed to dark green during the night. The balloon was now clinically in the duodenum; 81 ounces of dark green fluid were aspirated for the day, the pain becoming less.

At 10.30 a.m. on December 26 an X-ray film showed the balloon (which was never inflated owing to a known blockage in the tube) to be 18 inches beyond the duodenojejunal flexure. A non-residue diet of high protein and high vitamin content was commenced. The tube rapidly

passed down the bowel to a point in front of the fifth lumbar vertebra, where it remained for several days until operation on January 3, 1948 (Figure V).

During this migration the aspirated fluid was copious and fæculent; but when the tube tip became stationary the fluid became clearer greenish and less copious, remaining so until operation. However, attacks of pain, tenderness and borborygmi recurred, localized to an area to the left of the umbilicus. These appeared to be most severe when the caecostomy drainage diminished, and their subsidence was commonly followed by a profuse caecostomy discharge. Bedside skiagrams were taken on the mornings of December 29, 30 and 31, and January 1 and 2. A non-residue diet of high protein content was commenced on December 26. Valiant attempts were made by the patient to ingest adequate daily amounts, but without success, as on December 30 she vomited four ounces of greenish fluid containing white curds, the subsequent daily amounts of vomitus being two ounces, seven ounces and four ounces, the daily urinary output being 29 ounces on December 30, and 29, 15 and 16 ounces on the subsequent days.

On the morning of January 3, 1948, it was possible to make the following review of her case. Small-bowel obstruction from plastic adhesions had apparently followed the operation of December 16, 1947. Miller-Abbott intubation had resulted in rapid passage of the balloon into the duodenum and down to a high level block, notwithstanding that the balloon was never inflated. Satisfactory decompression of this length of bowel was indicated by the change of the fluid from fæculant to biliary, with a fall in the daily amount aspirated and the cessation of vomiting.

The existence of a closed-loop obstruction distal thereto was then indicated by persistence of recurrent attacks of colic, borborygmi and slight localized tender distension, and by the persistence in the daily skiagrams (Figure V) of a few distended small-bowel loops, which maintained their position in the centre of the abdomen while the tip of the tube remained fixed at one point, in spite of change in the aspirated fluid from fæcal to green and diminution in its amount after a few days. This loop was regarded as intermittently becoming unobstructed at its distal end, because daily X-ray examination showed the degree of distension to be diminishing (Figure V), and because of the intermittent discharge (associated with decrease in the colic and other symptoms) of very profuse amounts of fluid from the caecostomy. This loop was regarded as non-strangulated, as between the attacks of colic no pain was present, and the abdomen at the left of the umbilicus was relaxed and only slightly tender.

On December 29, because it was thought that a possibly formidable colonic resection was ahead of her, it was decided not to operate, but to maintain the decompression of the proximal part of the bowel, in the hope that the closed-loop obstruction might resolve, and to maintain nutrition intravenously and by the oral administration of a non-residue diet.

On January 3, 1948, a review of her progress showed that for the first four days after commencement of oral feeding on December 26 she had been able to take and retain increasing amounts of food by mouth, but that subsequently she vomited each day. The vomitus was biliary and contained milk curds; but the quantities of aspirated fluid (which was normal intestinal content) were much less than her intake. Nevertheless, it was concluded that there was insufficient length of bowel above the proximal obstruction to allow maintenance of adequate nutrition. Intravenous therapy had become difficult owing to thrombosis of the many veins that had been used.

Operation was therefore decided upon, not because of the unrelenting obstruction, but because of the problem of maintaining nutrition.

These conclusions were confirmed at operation the same day. A long loop of small bowel was found obstructed above by easily separated fine plastic adhesions to the pelvic brim, and below by similar adhesions to the left of the caecostomy, where the bowel was sharply kinked. This closed loop was moderately but not tensely distended, its wall being red and moderately thickened. Its mesentery was normal. The small bowel above the proximal block was pale, flaccid and completely decompressed, the enclosed Miller-Abbott tube tip lying a few inches above the proximal obstruction. There was no pus in the peritoneal cavity. After separation of the plastic adhesions causing the obstructions, the Miller-Abbott tube was threaded beyond the site of the distal obstruction and left *in situ*, not so much to provide decompression as to serve as an internal splint, in the hope that the inevitable subsequent adhesions would not form while the bowel was acutely kinked. After operation the caecostomy opening commenced discharging profusely, only a moderate amount of fluid being aspirated through the Miller-Abbott tube.

During the operation, blood transfusion was carried out. Within twenty-four hours severe hæmoglobinuria and hæmaturia and intense jaundice appeared and persisted until after the patient's death two days later. Subsequent investigation established that group IV Rh-positive blood had been given to a group IV Rh-negative recipient, who had received a previous blood transfusion.

At the post-mortem examination the abdomen was not distended. The peritoneal cavity contained clear fluid but no pus. The small bowel was not distended but was involved with some very recent adhesions. The large bowel was normal except for the carcinoma of the descending colon.

Several points in long-tube intubation are exemplified in this case, as follows: impaction of the balloon in the naso-pharynx, the rapid passage past the pylorus, and the speedy and complete migration of the uninflated balloon; the gradual emergence of the features characteristic of a closed-loop obstruction as the proximal part of the bowel was decompressed; the evidence against the presence of strangulation and for the occurrence of intermittent patency of the distal obstruction; the difficulty of maintaining nutrition in high jejunal block; the help at operation, in determining which is the proximal part of the bowel, afforded by palpation of the tube tip; and the use of a Miller-Abbott tube as an internal splint to prevent establishment of adhesions around acutely kinked bowel. Post-mortem examination suggested that, but for the tragic transfusion reaction, resolution of the acute obstructive problems would have progressed sufficiently to allow of colonic resection and treatment of residual small-bowel adhesions.

CASE VIII.—L.F., aged fifty-five years, weighing 17 stone, was admitted to hospital with severe distension, dehydration and peripheral circulatory failure following several days' abdominal colic, vomiting and constipation. Several milder attacks had followed hysterectomy some years before. The dehydration was corrected with serum, saline and glucose in water given intravenously. A Miller-Abbott tube was passed on May 8; she did not vomit again. The tube entered the upper part of the jejunum within thirty-six hours, but after passing in for 24 inches it remained localized to the upper left quadrant of the abdomen. Gas shadows rapidly disappeared from the serial X-ray films, the abdomen appearing homogeneously opaque. The aspirated fluid remained fæculant, the daily amount varying from 90 to 100 ounces.

Dull pain persisted in the lower and left aspects of the abdomen and back. Enemata gave negative results. A persisting close-loop obstruction, containing fluid only, appeared to be present, and laparotomy was performed on May 14, with the Miller-Abbott tube *in situ*.

The peritoneal cavity contained a little clear fluid. The ileum was adherent to the subumbilical incision in two places; but as the bowel above and below was dilated, hypertrophied and congested, and contained fluid, it appeared that the obstruction lay elsewhere. The proximal part of the bowel was identified by palpation of the tip of the Miller-Abbott tube several feet proximal to these adhesions.

None of the wall of this bowel was under tension, but the bowel was chronically dilated and contained a large quantity of fluid. Seventy ounces of this fluid (without any gas) were removed by enterotomy, evacuation through the Miller-Abbott tube appearing to be too time-consuming. This permitted examination of the pelvic cavity, where a short, tensely distended closed loop fixed at each end was found. The contents were fluid only. The wall was slightly cyanotic, but the peritoneum was healthy. Each end of the loop was firmly bound to the hysterectomy stump, and each adhesion was divided with difficulty. The affected loop was ten inches long, and at each adhesion stenosis from fibrous stricture was present. No areas of devitalization were present. The now flaccid loop had lost its cyanotic tinge. Resection of the loop and strictures with side-to-side anastomosis was performed, and the Miller-Abbott tube was threaded past the junction.

Continuous aspiration was carried out for the next four days, when, after two normal bowel actions and twenty-four hours of oral administration of fluid, the Miller-Abbott tube was clipped off. The tube was removed the next evening.

The clinical features and radiological appearances of a closed-loop obstruction, containing fluid only, were well

shown. The operation was difficult owing to the patient's obesity and to the deep situation of the major lesion; but all that was desirable could be done without subjecting the patient to undue risk. A similar procedure performed as an emergency without decompression or complete correction of fluid electrolyte deficiency must have proved both hazardous and technically difficult.

CASE IX.—Mrs. E.D., aged fifty-three years, had a barium meal examination performed for dyspepsia on June 24, 1946; this revealed small-bowel distension with multiple fluid levels. She was admitted to hospital two days later, a Miller-Abbott tube being passed to 67 centimetres and continuous suction applied.

Seventeen hours later, a skiagram showed the tube tip to be in the second part of the duodenum although still 67 centimetres from the nares. Four ounces of methylene blue solution were swallowed, and 110 seconds later the aspirated fluid seen in the glass Y-tube was coloured blue. The balloon was blown up and the tube was fed into the nostril. It rapidly migrated down the bowel. The quantities aspirated on successive days were four, 24, 12 and 13 ounces. The fluid was at first thin and green. On June 28, when the quantity had dropped to 12 ounces and the tube tip was well down the ileum, the fluid had gradually changed to thin fæculent and then to thicker light brown fluid of the consistency of thin cream, and identical in appearance with the fluid obtained from a caecostomy opening established for colonic obstruction. This fluid could not be aspirated with the suction apparatus, and only with difficulty and in small quantities with forcible syringe aspiration. Examination of a specimen excluded barium as a cause for this change. The following day the amount aspirated was 13 ounces, and at 10 p.m. vomiting of fæculent fluid commenced and persisted until laparotomy at 2 p.m. the following day (June 30).

A ring carcinoma of the transverse colon was discovered, with moderate dilatation and hypertrophy of the proximal part of the colon and considerable dilatation and hypertrophy of the ileum, which gradually diminished proximally. Caecostomy was performed and the Miller-Abbott tube removed. Subsequent treatment was on orthodox lines.

The difficulty of aspirating truly fæcal fluid is indicated. Sufficient tube to allow passage into the duodenum was provided by a length of 67 centimetres. Methylene blue taken orally appeared so rapidly in the aspirated fluid that this test alone was an unsatisfactory indication of the position of the tube tip.

CASE X.—G.D., aged forty-one years, a seaman, was admitted to hospital with pain of six hours' duration. Because of abnormal signs at his right lung base, and some increase in respiration rate and decrease in pulse rate after a few hours' observation, it was decided to observe him further. As the chest signs and respiration rate increased and the pulse rate fell to 84 per minute, and as the abdominal signs became less pronounced, he was transferred to the medical ward.

On the fourth day vomiting commenced and recurred repeatedly, 60 ounces of thin watery, yeasty fluid being vomited. His abdomen was slightly distended.

X-ray examination of the chest revealed gas under the right side of the diaphragm, with suggestion of a fluid level. The stomach appeared distended with fluid and with a large collection of gas in the fundus. Infiltration of the base of the right lung was apparent. At 8.15 p.m. intermittent aspiration via an indwelling Rehfuss tube was commenced. He did not vomit again. Eighteen ounces of fluid were aspirated to midnight. The following morning the abdomen was still distended. This distension increased during the day, being accompanied by a fair amount of abdominal pain. The evening temperature was 99·4° F., the pulse rate 80 per minute and the respiration rate 20 per minute. The amount of fluid aspirated for the day was 32 ounces.

He was transferred on this day (the fifth after his admission to hospital) to a surgical ward. The Rehfuss tube was replaced by a Miller-Abbott tube and continuous suction was applied. The tube was passed to the 70 centimetres mark and maintained there. The aspirated fluid amounted to 52 ounces for the day, being thin, watery, greyish, and of yeasty smell. X-ray examination five hours after the tube had been swallowed showed it to be still in the stomach, the small bowel distension indicating ileus. The report on an X-ray film taken twenty-four hours later stated that the tip of the tube appeared to be at the prepylorus in an atonic but collapsed stomach, and that a fair degree of small bowel distension was present. The evening report stated that he had had a comfortable day with two bowel actions. Both distension and pain were much less. The aspirated fluid for the day amounted to 50 ounces. The next day abdominal distension was much less. He passed several brown fluid stools flecked with mucus. A third X-ray film taken forty-eight hours after the passage of the Miller-Abbott tube, showed the tip well beyond the duodeno-jejunal flexure, with considerable diminution of the gaseous distension of the small bowel. The balloon of the Miller-Abbott tube was therefore blown up with 15 millilitres of water, instructions being given to thread it in through the nose gradually. For the day, the aspirated fluid amounted to 68 ounces, and was now bile-stained. The next day the abdomen felt much more comfortable. The administration of a non-residue diet and fluids by mouth was commenced. The aspirated fluid dropped to 26 ounces. The next day the Miller-Abbott tube was clipped off, and on the following day, as the patient had been taking food well without distress, the tube was removed. An X-ray film of the chest again revealed considerable gas under the right side of the diaphragm, but no fluid level. Convalescence was thereafter uneventful. The report on a barium meal examination five weeks after his admission to hospital read as follows:

> There is a persistent irregularity of the lesser curvature at the prepylorus. Stomach empties normally. Duodenal cap normal. Right diaphragm moves well. There is a large air space between this and the liver but it appears to be smaller than previously.

He was then discharged from hospital, well, for transfer to his home country, England.

On the morning of the fifth day after his admission to hospital, the clinical features and the X-ray film showing the stomach to be distended with fluid and gas suggested that the patient was suffering from a perforated ulcer which had spontaneously become sealed off; that there was some general peritonitis associated with established dilatation of the stomach, developing ileus and some subphrenic contamination; and that such peritonitis was apparently being satisfactorily managed by the patient. It was thought therefore that operative interference at that stage would be meddlesome, the treatment indicated being not for perforated ulcer *per se*, but for peritonitic ileus. The immediate indication appeared to be for intestinal decompression by means of Miller-Abbott intubation. This resulted in satisfactory decompression, and operative interference never became necessary.

Initial intermittent suction by a Rehfuss tube proved unsatisfactory, the daily amount of fluid aspirated being 18 and 32 ounces. Continuous Miller-Abbott suction resulted in complete decompression, the daily amounts aspirated being 52, 50, 68 and 26 ounces. The tube tip negotiated the pylorus and passed well into the jejunum with the balloon still collapsed.

CASE XI.—A.H., aged sixty-two years, with a history of 15 years' constipation, was admitted to hospital after six days' absolute constipation. For several months he had had recurrent attacks of severe central abdominal colic associated with borborygmi and occasional vomiting. His abdomen was tensely distended, but he responded rapidly to enemata. Ten days later X-ray examination after a barium enema disclosed an irregular filling defect at the hepatic flexure and pronounced small-bowel distension. On the following day colic and constipation recurred, and fæculent vomiting and distension followed. A Miller-Abbott tube was passed and continuous suction was commenced. Decompression was satisfactorily obtained, but the tube tip never left the fundus of the stomach until, on the fourth day, the tube was withdrawn (so that three inches projected through the cardia) and one millilitre of mercury was injected into the bag.

A skiagram nineteen hours later showed the tube tip to be in the duodenum. The bowels opened the following day and thereafter daily, and the oral administration of a non-residue diet with sulphathaladine was commenced.

Four days later the patient experienced a sudden attack of severe central abdominal colic, which gradually subsided after twenty minutes. He made no mention of this attack, and was thereafter well until laparotomy next day. A small carcinoma of the hepatic flexure was found. Proximal thereto the large bowel was dilated and hypertrophied, the ileo-caecal valve was widely open, and the terminal five feet of small bowel were much dilated and hypertrophied, this condition gradually decreasing proximally. Three feet

above the ileo-caecal valve was an intussusception seven inches long. The metal bucket of the Miller-Abbott tube was felt at the apex of the intussusceptum and projecting through it. The intussusception was reduced with extreme ease, there being no embarrassment whatever of the circulation of the intussusception or of the mesentery.

The recipient bowel was chronically dilated to a diameter of two and a half inches. The diameter of the intussusceptum was appreciably less. After reduction the balloon was felt to be only slightly distended and fitting loosely inside the bowel. Peritoneal secondary deposits being present, ileo-colostomy was performed, the Miller-Abbott tube being threaded to within six inches of the anastomosis.

A colonic obstruction with an incompetent ileo-caecal valve was satisfactorily decompressed by intestinal intubation, elective colectomy being thus permitted in the event of the condition's proving operable. Mercury injection caused rapid passage of the tube tip into the duodenum. Intussusception was precipitated by the balloon, but not because of excessive inflation. The variation in the diameter of the chronically dilated bowel contributed to the development of the intussusception, but at the same time eliminated strangulation.

It may be that cases of intussusception described elsewhere as a complication of intubation developed in bowel similarly chronically dilated and hypertrophied.

SUMMARY AND CONCLUSIONS.

Considerable difference of opinion exists as to the appropriate place of intubation suction decompression, enterostomy and laparotomy in the treatment of the obstructed bowel.

There is a tendency to regard intubation suction and enterostomy as alternative and competitive methods of decompressing the small bowel. The correct viewpoint is that intubation suction, enterostomy and laparotomy each have their place, to be used alone, successively or in conjunction according to definite indications.

Intubation suction, used according to such indications, has considerably decreased the necessity for enterostomy, and has rendered laparotomy unnecessary in some cases, and technically easier in all in which it had to be performed.

For intubation, a short gastric or duodenal tube and a long intestinal tube are minimum requirements. Of the latter, a tube constructed on the Honor-Smathers principle—that is, with a terminal bag which may be partly filled with both mercury and water—appears to be of greatest value.

When prolonged intubation suction is necessary, long-tube intestinal suction has material advantages over short-tube decompression. Decompression is more rapidly and efficiently attained, oral feeding simultaneously with suction may be instituted as soon as the tube tip is beyond the duodeno-jejunal flexure, and subsequent laparotomy is made considerably safer and technically easier, the presence of the tube, among other things, allowing ready identification of the proximal part of the bowel and direct decompression of bowel suture lines.

The constructional details and mode of use of a modification of Wangensteen's suction apparatus, possessing several advantages, are described. A list of instructions to the nursing staff for the management of the apparatus is detailed.

In all cases of small-bowel obstruction, with one exception, long-tube intubation with continuous suction should be commenced without delay, the necessity for operative treatment in addition depending, commonly in this order, on the presence of strangulation, the failure of attainment of satisfactory decompression, the inability to maintain adequate nutrition, and the failure of the obstruction to resolve. Each of these factors is fully discussed.

Failure of attainment of decompression is due in most, if not all, cases to the presence of a closed-loop obstruction, which, if persistent, requires either enterostomy or direct release of the obstruction, continuance of proximal intubation constituting an essential part of the post-operative management.

It is probable that enterostomy as a method of treatment of intestinal obstruction has proved of variable value in the past, not because it was essentially a poor method, but because it was not appreciated that it was often necessary to combine enterostomy of a closed loop with intubation decompression of the bowel above the proximal block of such a loop.

In large-bowel obstruction intestinal intubation decompression may be worthy of trial in two classes of case. Firstly, when obstruction is early and colonic distension is slight, suction, by preventing further passage of small-bowel contents into the colon, may prevent further increase in colonic distension for a sufficient period to allow conservative measures to succeed in reestablishing colonic patency. Secondly, when colonic obstruction is associated with an incompetent ileo-caecal valve, the caecal wall is hypertrophied and the intraluminal pressure never becomes of high degree. Colonic decompression is thus not so urgently required.

Considerable advantages are associated with successful intubation decompression in these cases. The necessity for colostomy or caecostomy, which might prove a technical embarrassment to subsequent colectomy, is avoided; and if the tube is left in position during the pre-operative preparation, it will provide useful decompression of the suture line of ileo-colostomy after right colectomy, or of the small bowel after left colectomy.

In respect to intubation, intestinal obstructions may be divided into three groups. Group I comprises those requiring early operation, intubation being used as an adjunct to treatment. It includes strangulating small-bowel obstructions and all large-bowel obstructions. Group II consists of those cases in which prolonged intubation is expected, in most cases, to result in resolution of the obstruction. These are obstructions of the early post-operative period, and those accompanying peritonitis. Group III includes the remaining non-strangulating small-bowel obstructions. In these, long-tube suction is used with the knowledge that at the end of a variable period of suction operation will probably be performed. It is used because of the striking contrast between the conditions found at such elective operation performed on the hydrated, nourished patient, with decompressed bowel, and those found in the dehydrated and starved patient with abdominal distension subjected to immediate operation. This contrast is so striking that it is held that the advantages far outweigh the danger of overlooking a serious strangulation. At operation the patient's general condition is usually excellent; in most cases the bowel is collapsed and may be readily packed away, and the obstructing lesion is usually readily accessible and found to have a healthy blood supply. Radical procedures are thus possible, which, even if practicable at operation in the presence of unrelieved distension, would have been associated with a post-operative period fraught with danger. In all cases the tube, if possible, is manipulated to any suture line and left *in situ* for a few days. In this group, coincident disease may warrant a trial of prolonged suction, in the hope that the obstruction will, even if only temporarily, resolve as in Group II.

There is a place for a similar attitude in certain cases in which the patient is known to have widespread "matting" of bowel loops by old adhesions and has had one or more operations for adhesions.

Details are given of the management of long intestinal tube intubation in obstruction, these including the passage of the tube, the determination of its passage through the pylorus, the changes occurring during migration of the tube and during the establishment of decompression, the phenomena associated with maintenance of established decompression, the criteria of resolution of the obstruction, the radiological appearances at various stages, and the maintenance of hydration and nutrition throughout intubation.

With these methods it has become apparent that if the tube tip comes to lie in the gastric fundus, it is apt to remain there for an inordinate period; but if it can be made by various manœuvres to lie in the prepylorus it

will pass within a reasonable period through the pylorus and down to the most proximal block.

The factors leading to the passage of the tip into the fundus and its retention therein are discussed. It appears that the most satisfactory design of tube is one of the Honor-Smathers type—that is, a tube with a soft terminal bag hanging from the end of a double lumen tube allowing of alteration of the contents of the bag during intubation.

A technique of intubation based on the above discussion and by means of such a tube is described.

Eleven cases, two with fatal terminations, have been recorded to illustrate the advantages, disadvantages and technical details of intubation.

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Reports of Cases.

REPORT OF TWO CASES OF HERPES ZOSTER TREATED WITH "CHLOROMYCETIN".

By N. F. BABBAGE, F.R.C.S.E.,
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Case I.

MRS. N.B., aged sixty-three years, was first examined on March 21, 1950, complaining of pain in the left sde of her neck and face. She was debilitated and on examination was found to have mild general pharyngitis, but no other cause for her neuralgic pain was discovered. When she was next examined, on March 27, the cause was obvious; she had early well-developed *herpes zoster* in the distribution of the second and third cervical segments, some vesicles extending into her hair posteriorly. It was very painful. The expense of "Chloromycetin" was explained to her, but as a near neighbour was still suffering from post-herpetic neuralgia some two years after an attack, she was willing to try. Accordingly, on March 29, the herpes being well developed, she was given two capsules of "Chloromycetin", the dose to be repeated in two hours, after which one capsule was to be taken every four hours (24 capsules in all). Within four days the rash was reduced to a number of dry scales. On April 12 the rash was completely healed and no neuralgia was left.

Case II.

Mr. A.T., aged twenty-four years, was first examined on August 24, 1950; he gave a history of a rash on the abdomen and back present for three days. Examination revealed fairly severe *herpes zoster* in the distribution of the tenth and eleventh left thoracic segments. It was painful. Blebs were breaking and it was keeping him awake at night.

Again the proposition of cost was put to him, and he elected to pay for "Chloromycetin" treatment. Accordingly the same day he commenced with a dose of three capsules, followed by two capsules in two hours, then one every four hours—a total of 24 capsules. On August 26 there was an amazing improvement. The vesicles were flat and drying. On August 30 the condition was nearly healed; many of the scales were already separating. Healing was complete within a further two days.

Comment.

The response was so rapid that I intend at the next opportunity to use 12 capsules only and await results. Another interesting point is that if herpes is so sensitive, might not poliomyelitis, which is closely akin to it, also respond? There is no report that I can find, or that the makers can supply, of any trial of the use of "Chloromycetin" in poliomyelitis.

Reviews.

DERMATOLOGY FOR GENERAL PRACTITIONERS.

"MODERN PRACTICE IN DERMATOLOGY", edited by G. B. Mitchell-Heggs, a further addition to the "Modern Practice Series" (Butterworth and Company), is a text-book of skin diseases written primarily for general practitioners.[1] It is a symposium by forty-one authors from different teaching schools of the Commonwealth, and its fifty chapters thus show a variety of styles and methods of presentation which makes for interesting reading.

Such a collection of modern opinion is bound to contain much that is debatable, but the style throughout the book is dogmatic, and the subject matter is presented in an essentially practical manner, so that the work fulfils the aim of the editor to produce a blend between a manual and a text-book.

The editor has wisely refrained from attempting to classify the skin disorders under consideration in the usual way, but has adopted a practical grouping based on both ætiological and regional considerations. The method of grouping has, however, led to rather too much repetition, a number of subjects being discussed more than once. There are also several omissions. For instance, Besnier's prurigo (atopic dermatitis) is discussed in the chapter on psychosomatic disorders, but a description of the clinical features, diagnosis, prognosis, and treatment of this important condition is not to be found.

There are 319 illustrations in black and white and seven colour plates. All these are well chosen and exceptionally clear. The paper and binding are excellent, and the index is particularly good.

This is a book which will appeal to all practitioners who are interested in dermatology.

A STORY OF NUTRITIONAL RESEARCH.

IN 1913 fat-soluble vitamins were discovered by McCollum and Davis in the United States, and the Medical Research Committee was established in Great Britain. These two apparently unconnected events were together destined to have a profound effect on the development of our knowledge of nutritional requirements.

One of the first problems to which the newly formed Medical Research Committee (now Council) directed its attention was the ætiology of rickets, then a prevalent disease in Europe and North America. The investigation of this problem was entrusted to Edward Mellanby. Through the delays and frustrations of two World Wars Mellanby has continued these investigations to the present day. In 1941 he was invited to give a course of the Abraham Flexner Lectures at Vanderbilt University. Owing to the exigencies of World War II these lectures were not delivered until 1947. The lectures have now been published in "A Story of Nutritional Research".[2] In it Sir Edward describes, with much of the appropriate detail, the various ramifications of the problem into which he and his collaborators were led in the course of their investigations.

Early work was hampered by ignorance of the multiple nature of the fat-soluble substance which McCollum and Davis had named vitamin *A*. The problem had early been recognized as being primarily concerned with calcium metabolism. This fact was for a time obscured by the arresting effects of vitamin *A* deficiency on the nervous system. Such effects were shown later to be secondary to the mechanical effects of abnormal growth of bone contiguous to nerve tissue. The elucidation of these complicated effects and the demonstration that rickets was produced by deficiency of another group of fat-soluble vitamins, later named *D*, and the effect of this on the absorption and utilization of calcium, make a fascinating story. The help and inspiration of Lady Mellanby, who at the time was carrying on her own well-known investigation on dental caries, was undoubtedly of considerable importance.

The work led naturally to an investigation of supplies of calcium and of phosphoric acid available to man in his various foodstuffs for the formation of bone. A surprising result of this part of the work was the discovery of the decalcifying effect of cereals. Oatmeal was found to be particularly bad in this respect. The author had the temerity to announce this discovery at the Glasgow meeting of the British Association for the Advancement of Science in 1922. The local reaction was immediate and violent. Repercussions were world-wide. The effect was at first attributed to some sort of evil vitamin for which the author proposed the name of toxamin. It was not long, however, before the offending substance was found to be the well-known plant constituent phytic acid, which produced its effect by forming insoluble salts of calcium.

Certain aspects of the action of vitamin *A* and *D* have not been dealt with by the author. Although his story is as yet not all told, it is one of the most complete accounts of a long, tedious and successful series of investigations which has been published. The book is excellently produced. It has numerous illustrations, graphs and tables. Complete author and subject indices and numerous references are included.

MAMMALS OF VICTORIA.

"THE MAMMALS OF VICTORIA", by C. W. Brazenor, is a splendid handbook, a type of which we have few.[1] The author, a well-known authority, is the mammalogist of the National Museum of Victoria—the institution responsible for its publication. Other handbooks in this series are forecast. The information is concise, simply phrased and naturally reliable. The line drawings by G. J. Browning are a feature and these are so placed with the relevant text that one may read and see simultaneously.

There are, however, some points which merit attention in later editions, or the handbooks which are to follow. It is understandable that the work be restricted to those species occurring in Victoria, but surely the Victorian reader would be interested to know which species range northward to Cape York and which to Cape Leeuwin in the west. Lack of space cannot be pleaded for this omission, for unused space abounds. The chapter on dentition has, no doubt, been placed last purposely lest its technical nature should dismay that ubiquitous individual, the man in the street, for whom the series is mainly intended. Yet it should have preceded the index and the matter should have been entered therein.

Despite these comments the book is very useful, and it is another illustration of the modern trends of museums in popular education.

AN ATLAS OF ANATOMY.

THE second volume of Woerdeman's atlas[2] (the first was reviewed recently in these columns) has just appeared, thus, we understand, completing a work which was originally designed for three volumes. The present volume covers splanchnology, angeiology, the nervous system and sense organs. As in Volume I, there is no text. The author mentions the difficulty of deciding upon a limit to the detail supplied—in this case what can be discerned with a hand lens. The figures are excellently drawn and reproduced, the nomenclature is in Latin. A few minor criticisms occur. Some of the illustrations adhere to a purely formal pattern—in the blood vascular systems, for example, there is no hint of variations. It would be useful to know the ages of the three fœtal brains depicted in figures 422-424. The author is inconsistent in the matter of detail. He omits reference to the deep blood supply to the brain, yet includes a number of diagrams illustrating the major pathways and nuclei within the central nervous system. These and other points do not, however, detract from the fact that the atlas will prove an exceedingly valuable adjunct to any course in anatomy.

[1] "Modern Practice in Dermatology, 1950", edited by G. B. Mitchell-Heggs, O.B.E., M.D., F.R.C.P.; 1950. London and Australia: Butterworth and Company (Publishers), Limited. 6½" × 9½", pp. 866, with illustrations. Price: 82s.

[2] "A Story of Nutritional Research: The Effect of Some Dieting Factors on Bones and the Nervous System", by Sir Edward Mellanby, G.B.E., M.D., Sc.D., F.R.S.; 1950. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 8" × 5½", pp. 462, with 73 illustrations. Price: 53s. 9d.

[1] "The Mammals of Victoria: and the Dental Characteristics of Monotremes and Australian Marsupials", by C. W. Brazenor; 1950. Melbourne: Brown, Prior, Anderson Proprietary, Limited. 8½" × 5½", pp. 130, with illustrations. Price: 7s. 6d.

[2] "Atlas of Human Anatomy: Descriptive and Regional", by M. W. Woerdeman, M.D., F.R.N.A.Sc.; Volume II, Splanchnology—Angeiology Nervous System—Organs of Sense. London and Australia: Butterworth and Company (Publishers), Limited. Amsterdam: Wetenschappelijke Uitgeverij N.V. 10" × 6½", pp. 704, with 642 illustrations.

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CANCELLATION OF MEETINGS IN SOUTH AFRICA.

WHEN members of the Australian Branches of the British Medical Association receive the *British Medical Journal* of February 17, 1951, they will read with disappointment the decision of the Council of the Parent Body to cancel acceptance of the invitation from the Medical Association of South Africa to hold a joint meeting at Johannesburg in 1951. Without exception members of the Australian Branches will approve the decision and applaud the dignified way in which it has been made. It will be remembered that the meeting of the Representative Body was to have been held at London and that the annual meeting, the scientific side of the Association's annual activities, was to be held at Johannesburg in conjunction with a meeting of the Medical Association of South Africa, a body in affiliation with the British Medical Association. This was the kind of arrangement that was adopted in 1935 when the annual meeting of the whole Association was held at Melbourne. At the last meeting of the Representative Body in 1950 consideration was given to a recommendation of the Council that the acceptance of the invitation to go to South Africa should be withdrawn. After a long discussion the Council's recommendation was rejected. Since then the Medical Association of South Africa, in view of recent political developments in that country, sought from the Minister of the Interior of the Union an assurance that no difficulty would be placed in the way of any member of the British Medical Association who wished to attend the meeting—that entry into the Union for this purpose would be easy. The Minister replied that he could not give this assurance. Members of the British Medical Association come from every country belonging to the British Commonwealth of Nations, and the Council could not continue in a course of action which might expose members to affront or discourtesy or other unpleasantness. Dr. A. W. S. Sichel, who was to have been installed as President of the British Medical Association at Johannesburg, will be invited to go to London to be installed as President and to give a president's address.

A logical sequence of the Council's action has been the cancellation of the British Commonwealth Medical Conference which was to have been held at Johannesburg just before the joint meeting. Many will regret that this has been necessary, but all will agree that no other course of action was possible. The Federal Council of the British Medical Association in Australia had resolved to be represented. The next conference will probably be held in India in 1952, but more will be heard of that possibility at a later date. In the meantime the members of the British Medical Association in Australia will affirm their feelings of kinship and cordiality towards members of the Association in all countries and towards members of associations affiliated with it in different parts of the British Commonwealth.

FORTITUDE.

THE doctor owes much to his patient, without whose cooperation, confidence and courage the armaments of science would be of less avail. It is often remarked with some air of surprise that children "make good patients": that is true, but once pain and fright are conquered children are not tormented by those dreads of the older folk, who see only too clearly what evils may befall. E. C. Temple Smith has recently given a striking example of the value of courage in a patient who, with one eye lost, and the other temporarily blind, did not know if he would regain his sight or remain like Samson, "dark amid the blaze of noon". Ophthalmologists know well, too, that peculiar trial of the old, the ordeal of cataract extraction; not so much the surgical procedure, as the after days of waiting. All practitioners, in whatever field they work, are sympathetic of like situations, and know that the strain on the moral courage of the ill is more severe in those of livelier imagination and quick sense of realism. Rudyard Kipling in one of his tales of India described fear as it affects animals, and endowing them with speech, exposed their strengths and weaknesses. The mule that nonchalantly carried screw-guns over slippery mountain ridges, and learnt to keep out of sight of unseen enemies, was afraid of bullocks and panicking camels. The troop-horse that was not dismayed by a charge of knife-armed fanatics was scared at what he could not see at night. The camels that sat down placidly in a square, and let men fire over their backs, would break up a whole camp through dream fears in the dark. The gun bullocks that would graze unconcernedly during action were afraid of white men. The elephants, themselves disturbing to the horses and mules, would refuse to approach shell-fire nearer than the point where their intelligence told them there was danger, and yet, unreasoning and unaccountable, they were afraid of small animals. These inconsistencies of courage and fear find their parallel in the human subject. What can we do, then, to help those who have to face physical and mental ordeals?

Horace dismissed the problem in a famous aphorism,

> *Aequam memento rebus in arduis*
> *Servare mentem.*

Bertrand Russell says that the experience of a long life has taught him that Latin tags always express falsehood. This is a hard saying, yet it is perhaps permissible to try to understand what this Horatian tag means in modern terms. How can we "remember" to keep a calm mind in

times of stress, and in this sense, what is "mind"? We are exhorted to remember; we must keep memory fresh. Yet in trying to understand what memory is we may find it hard to accept the mechanisms of the behaviourist psychologist. Memory, however gained as a human asset, must have some accompaniment of imagination, and in the strong excitement of trouble or fear, belief, so often lacking in imagination, must exist, for without faith what are we but particles in a void? Let us then accept an expansion of Horace's "*memento*", and exhort the man in trouble to use mental associations, imagination and experience, and to bring the past into the present, and even the future, so that faith and hope may come to his aid. Our analysed Horace seems to take a physiological turn; at least it is fair to ask just how we reach that admirable equanimity of mind, and what nervous functions are concerned.

Long ago Hughlings Jackson realized the philosophical implications to a neurologist of consciousness, and recognized its degrees. He probably would have agreed with William James that consciousness represents not an entity but a function. This function must surely in some sense involve the whole of our nervous system. By experience, which implies integration of a complex receptor system, we connect other mental events with one particular event, but we cannot isolate parts of our brain from its effects. The more primitive diencephalon may sway the resultant events more than that prized possession of the higher orders, the neopallium. Yet, though we show the physical stigmata of fear, tension, pallor, rapidly beating heart, the implied flight reaction may be suppressed below the level of action, and the faint heart literally becomes strong. Charles Lamb describes his hag-ridden infancy, with its terrors of the night, and apologizes for the tame and prosaic dreams of his later life. Was it an accident that this timid youth, burdened with the lifelong horror of a mad sister discovered in grim and fatal acts of violence, conquered his fears? On the one hand was Lamb, pathetically conducting his sister to an asylum whenever her cyclic mania beset her, both with tears in their eyes, and bearing a mediæval strait-jacket. On the other hand is Lamb of the "Essays of Elia", master of a light touch of literary sincerity, a sober civil servant, and interestingly enough, writing of puns and punning to a friend in Sydney.

Philosophy and physiology may not teach us how and where this mystic change is wrought in our "mind", whatever that is, but we must agree with Horace as a poet, and perhaps the poets know best. The doctors cannot well explain, but they know, too, and frankly give homage to their strongest allies, their patients.

Current Comment.

A FACTOR INHIBITING BLOOD MATURATION IN THE MARROW IN PERNICIOUS ANÆMIA.

THE application of methods of culture of the cells of the bone has yielded some interesting and perhaps important results. Strong evidence has already been produced by some workers that there is an inhibitory factor in the serum of patients with pernicious anæmia during a phase of relapse. This inhibitory effect *in vitro* increases in intensity as the concentration of pernicious anæmia serum is increased, though the same phenomenon has not been noted when normal serum is used in the experiments. Further work has been done on this subject, which sheds light on what is apparently a much more complex process than was once thought; in fact the work now planned is designed to reveal the nature of the inhibitory factor, and to help to explain the curiously contradictory relations between the hæmatological and neurological symptoms and signs of the Addisonian type of anæmia, especially when the modern highly refined methods of treatment are used.

R. B. Thompson in a recent important communication has described[1] the results of culture experiments on marrow cells aspirated from patients suffering from pernicious anæmia and kept in varying proportions of plasma from normal subjects and those with pernicious anæmia. Differential counts were made of the red cells to ascertain their degree of maturation, graded into four types according to a definite histological pattern. Either plasma or serum could be used; the former required the addition of a small amount of heparin, not sufficient to inhibit growth. It was found that good evidence existed of the presence of an agent in the plasma in pernicious anæmia which *in vitro* had the power of inhibiting the maturation of primitive marrow cells of patients in a state of relapse. This agent also had the power of annulling the maturing effect of the blood of normal persons. Thompson suggests that there is in the serum and plasma of patients with pernicious anæmia an agent capable of inhibiting maturation of erythroblasts in the body as well as *in vitro*. He further suggests that the mechanism of the inhibition is by competition, whereby an enzyme system is blocked from supplying a necessary nuclear constituent of marrow cells.

L. G. Lajtha has presented work along the same lines.[2] He cultured 31 cell suspensions from human marrows, and observed the degree of progressive change in the erythroblasts. This he divides into two phases of development: "ripening", meaning the transformation of megaloblasts to normoblasts, and "maturation", meaning the development of later cell forms of both normoblasts and megaloblasts. Experiments were designed to show the effect on the cultured cells of normal and "pernicious" serum, with varying concentrations, the effect of the pernicious anæmia serum on normal marrow cells, its sensitivity to heat, the presence of the factor in the cerebro-spinal fluid, the effect of adding known antipernicious-anæmia factors. The evidence again shows the presence of an inhibitory factor, but, as Lajtha points out, it may not be in nature abnormal though it may suffer eclipse by a hæmopoietic factor. He finds that this factor will transform the normoblasts of originally normoblastic marrows into megaloblasts. It is thermostable, and is also present in the cerebro-spinal fluid of patients in a state of relapse. Folic acid will neutralize the effect of this inhibitory factor *in vitro*; so, too, will liver extract, but vitamin B_{12} has no such effect.

Lajtha also makes suggestions as to the method by which the hæmopoietic changes of pernicious anæmia are brought about, and reversed by treatment. He thinks it possible that the vitamin B_{12} molecule acts as a coenzyme to a necessary enzyme system which is changed by the body to the hæmopoietic factor. Lack of this vitamin, due to deficient absorption from the intestine, may then cause metabolic disturbances in the nervous system, and an inhibition of the normal concentrations of folic acid, which could be overcome by mass action. All this is too complicated for a simple and condensed exposition, and we cannot help recalling that pernicious anæmia has caused the demise of many promising hypotheses. This disease, however, is one of the few conditions in which steadily improved methods of treatment have been the gift of the research workers, even though they found the subject was becoming more and more complex.

Perhaps now the riddles are nearing their ultimate solution, if we can say that there is such a thing in the realm of medicine.

[1] *Clinical Science*, 1950, Volume IX, Number 3.

[2] *Ibidem.*

THE EFFECT OF TERRAMYCIN ON THE INTESTINAL BACTERIAL FLORA.

A NUMBER of substances have been used with the object of controlling the bacterial flora of the bowel, chiefly with the purpose of increasing the margin of safety in surgical procedures involving the lower parts of the intestinal tract. Previous work at the Mayo Clinic has shown that one of the recently introduced antibiotics, terramycin, can effectively be given by mouth, and that it is excreted in the fæces in high concentration. This has led Joseph M. Di Caprio and Lowell A. Rantz, of the Department of Medicine in the Stanford University, to investigate further the degree of control exercised by this substance on the bacterial population of the intestine.[1] They carried out cultural studies designed to determine the quantitative and qualitative data concerning these bacteria, and then to ascertain their sensitivity *in vitro* to penicillin, dihydrostreptomycin and terramycin. Cultures were made of fresh specimens of stools on plain blood agar and MacConkey's medium, and also on blood agar containing stated concentrations of the antibiotics under review. Only a small series was examined, consisting of seven patients, who were suffering from such diverse conditions as regional ileitis, amœbiasis, recto-vaginal fistula, non-specific ulcerative colitis, rectal ulceration of doubtful ætiology, and as a control, *herpes zoster*. All the patients were treated with terramycin, given in dosage of 750 milligrammes orally every six hours, for a period of days from two upwards, according to the nature of the condition. Daily examinations of the stools were carried out. The most striking finding was that the ordinary ærobic bacteria of the bowel disappeared or were reduced to a total concentration of less than 2000 organisms per millilitre of wet stool. The organisms found included members of the coliform group, non-hæmolytic streptococci and the *Streptococcus fæcalis*. Most of the bacteria were found to be resistant to penicillin and sensitive to dihydrostreptomycin, and all were sensitive to terramycin, with the exception of Proteus and the yeasts. The disappearance of these bacteria was observed within forty-eight hours of the beginning of administration of terramycin. In several cases resistant strains were found after a variable number of days; some of these had not been previously found, or had been observed to be sensitive earlier. Obviously the series is too small to enable sure conclusions to be drawn. However, it is striking that the normal ærobic flora of the bowel were virtually eliminated after two days' treatment by terramycin, that is, with the oral administration of three grammes of the drug. Other workers have found that streptomycin and aureomycin had a similar effect, and so far it is suggested that their ability to control intestinal bacteria is greater than that of those members of the sulphonamide series which have been used for this purpose.

Di Caprio and Rantz consider that their results show that terramycin compares favourably with other agents, and further suggest that it might be advisable to use this antibiotic with another, such as streptomycin. Though terramycin has been reported as less toxic than some other preparations, a high incidence of gastro-intestinal symptoms was observed in this series. Epigastric discomfort, anorexia, nausea and vomiting were common, although they did not interfere with therapy. If a short pre-operative period of medication will serve the purpose of the surgeon who wishes to operate with greater safety this might not be a serious drawback, but the continuance of therapy after operation might have definite disadvantages. No toxic effects were observed by examination of the blood or the urine, though a few red cells were found in the urine of one patient on the twelfth and last day of treatment. This preliminary account suggests a possible use for terramycin in surgery of the intestinal tract, but it seems fair to state that conclusions are tentative at present. It appears that the great advantage of being able to administer some of these substances by mouth has an occasional drawback, the occurrence of irritative local symptoms; whether this is of any real significance can only be determined with the passage of time.

[1] *A.M.A. Archives of Internal Medicine*, November, 1950.

THE PATHOGENESIS OF POLIOMYELITIS.

IN view of the present widespread epidemic of poliomyelitis, and the feeling among practitioners that they are seeing examples of unusual disease which is not poliomyelitis as they understand it, but yet bears some of the marks of a virus infection, a review of the recent advances in the pathogenesis of the disease is timely.

Such a review is presented by Claus W. Jungeblut.[1] He points out that it is now known quite well that non-paralytic cases of poliomyelitis far outnumber the paralytic, and indeed it seems reasonable to say that the non-paralytic form of the disease is the normal and typical response of the human being to the infection, the occurrence of paralysis being unusual and analogous to the occurrence of encephalitis in measles. Virus has been obtained from the stools and naso-pharyngeal washings of non-paralysed patients. When the cynomolgus monkey was used instead of the rhesus, subclinical disease was produced, and the virus was obtained from stools and the walls of the pharynx and from abdominal viscera of infected animals. Oral infection in the chimpanzee produced subclinical poliomyelitis with prolonged excretion of the virus in the stool. It has been found that the virus of poliomyelitis can produce lesions in the peripheral neuromuscular apparatus and not only in the neuronal tissues, and that it is capable of living and multiplying in tissues outside the central nervous system. Lesions in the myo-neural junction have been found, with rapid destruction of motor end-plates, but there is no proof that these are primary. Lesions in muscle, apart from atrophy due to neuronal destruction, have been found. On one occasion virus obtained from a muscle biopsy produced typical disease in a cynamolgus monkey. Myocarditis has been found, consisting of focal necrosis of individual heart muscle fibres and varying degrees of inflammatory cell response. Tachycardia is sometimes a clinical feature of the disease, and the electrocardiographic findings have been reported as abnormal in 32 of a series of 226 cases, with significant correlation between severity of illness and occurrence of cardiac anomaly. In some subpassages of virus obtained from heart muscle, myocarditis as well as cord lesions resulted, suggesting the possibility that heart damage may be the result of infection with a certain strain of virus. It may be the cause of death in some severe cases, apparently from cardiac failure while respiration is being mechanically maintained. Many attempts to demonstrate viræmia have been made. In one series the virus was obtained from the blood stream of one out of 111 patients, only 20 of whom, however, were seen on the first day of the illness. This result does not exclude early and transient viræmia. The discovery of a number of varying strains of virus able to produce poliomyelitis-like disease has added to the complexity of the picture, but has opened the way for a more satisfying understanding of it.

Jungeblut's view seems reasonable and in keeping with the disease as we are seeing it. He summarizes his conception of the disease as probably following a sequence such as this. The first stage consists of entrance of the virus through the alimentary tract, its penetration of the intestinal mucosa and growth in the gut wall. At this stage the disease is symptomless. The second stage may consist of transient blood-stream spread of the virus and its fixation in heart and skeletal muscle. In this stage one may see symptoms of slight fever, malaise and scattered peripheral discomfort. It is only in a small proportion of cases that the infection spreads further to produce clinical poliomyelitis as we know it, and in them Jungeblut suggests that excessive growth and activity of the virus allow its passage across myo-neural synapse and along peripheral nerve to the central nervous system.

[1] *The Journal of Pediatrics*, July, 1950.

Abstracts from Medical Literature.

OPHTHALMOLOGY.

Cortical Potential Changes in Amblyopia ex Anopsia.

DALLAS DYER AND EDWARD BIERMAN (*American Journal of Ophthalmology*, July, 1950) have studied the electro-encephalographic findings in 33 cases of *amblyopia ex anopsia* associated with strabismus; 28 of the patients affected were between the ages of four and fourteen years and five between the ages of seventeen and thirty years. Of the 28 children, 24 had abnormal cortical wave patterns. In the tracings for the five adults only one showed abnormal waves, and these occurred after hyperventilation. The authors found a widespread active cortical potential disturbance in the majority of cases in children. In adults, however, some change occurs, for abnormal waves disappear in the majority of cases. The electro-encephalographic tracings in children are similar to those found in convulsive states. The authors suggest that the abnormal cortical excitation may account for some of the behaviour problems in children with suppression amblyopia. They consider that their results support the theory of active cortical suppression of the form sense in children.

Surgical Treatment for Conical Cornea and Astigmatism.

TUTOMU SATO (*American Journal of Ophthalmology*, June, 1950) describes his method of treatment of conical cornea, astigmatism, keratectasia and deformities caused by post-inflammatory partial expansion of the cornea. An incision is made in the posterior surface of the cornea by passing a special lancet designed by the author into the anterior chamber. The author claims the procedure to be both safe and effective. He describes in detail the procedure to be adopted for conical cornea and astigmatism. Between 1938 and 1943 he performed the operation on 200 patients with conical cornea. In almost all cases there was improvement of vision and refraction.

The Practical Importance of Abnormal Retinal Correspondence.

T. a'B. TRAVERS (*Transactions of the American Academy of Ophthalmology and Otolaryngology*, May, 1950) discusses the problem of abnormal retinal correspondence. He states that if the patient is cooperative, diagnosis of abnormal retinal correspondence is simple, by means of examination on a synoptophore with two simultaneous macular perception slides. If the patient is too young to cooperate, correspondence can be guessed at. The correspondence is likely to be abnormal if the angle of squint is over 30° and if the age of onset of squint is under two years. The likelihood of abnormal retinal correspondence increases directly with the angle of squint and inversely with the age of onset of the squint. It is unlikely if the onset of the squint is after four years of age. It is unlikely if the angle of squint is under 20° except in divergent squint. It is more likely if the squint is constant and less likely if the squint is variable in degree. Four methods suggested for the treatment of abnormal correspondence are alternating occlusion, prescription of prisms, orthoptic exercises and corrective operation. However, the only treatment worth while is operation. If performed before the age of three years there is a reasonable chance that normal binocular vision will develop. Once a diagnosis of abnormal retinal correspondence is made, operation is desirable.

The Dupuy-Dutemps Dacryocysto-rhinostomy.

ALTON V. HALLUM (*American Journal of Ophthalmology*, September, 1949) concludes from his experience with the Dupuy-Dutemps dacryocysto-rhinostomy that there should be relief from tearing and muco-purulent discharge in 95% of cases. The operation is indicated in stricture of the naso-lachrymal duct or any portion of the lachrymal sac, or in cases of absence of the lachrymal sac, provided the lower punctum and lower canaliculus are normal. Dilatation of the lachrymal obstruction should be attempted in all cases, but if the passage of a number 2 or 3 Bowman probe into the nose does not relieve the obstruction after two or three weekly probings, it is useless to continue. Repeated probings that progressively dilate the lower punctum are contraindicated, since they will cause tearing after a subsequent dacrycystorhinostomy. The author describes the operative technique in detail. He recommends that after the nasal mucous membrane and the medial wall of the lachrymal sac have been exposed, a vertical incision should be made in each and then posterior and anterior flaps sutured, an epithelium-lined passageway being thus produced from the lachrymal sac into the nose. Post-operative irrigations are not necessary.

Fibrin Closure in Eye Surgery.

ARNO E. TOWN AND DAVID NARDOFF (*American Journal of Ophthalmology*, June, 1950) describe the use of fibrin for the sealing of ocular wounds. They state that the principal aim of fibrin closure is to prevent the complications incident to a leaky wound by providing an even and quick-healing incision. The traumatism caused by the introduction of corneo-scleral sutures and their post-operative removal is averted. In addition perfect closure will prevent epithelial invasion of the anterior chamber. The authors describe the preparation of the plasma and its use in sealing the wound after cataract extraction. The authors now use fibrin closure as a routine measure, having discarded sutures.

Incision and Closure of the Wound in Cataract Operations.

F. A. DAVIS (*Archives of Ophthalmology*, August, 1950) has studied the results obtained in intracapsular cataract extraction with a keratome-scissors incision and with a Graefe knife incision. Results in 257 cases with a keratome-scissors incision are compared with those in 202 cases with a Graefe knife incision. Deep sutures were employed, and most extractions were combined with iridectomy. Analysis of the complications which occurred shows that loss of vitreous and iris prolapse were about equal in both techniques. The incidence of irido-cyclitis was greater after the keratome-scissors incision. Post-operative hyphæmia was three times more frequent when the keratome-scissors incision was used. The incidence of secondary glaucoma showed a significant increase after the keratome-scissors incision as compared with the Graefe knife section. Delayed restoration and late loss of the anterior chamber and incomplete closure of the wound were more frequent in the keratome-scissors incision. Chorioidal detachment was observed in 47 cases in which keratome-scissors were used as against five cases in which the knife was used. The author believes that the trauma produced by the use of scissors is the main contributing factor in delayed wound closure, subsequent leakage and the various complications which may follow this. He concludes that the keratome-scissors incision has been useful when the chamber is very shallow; however, complications which have followed this technique have led to its abandonment as a routine procedure.

Choice of Operation in Acute Glaucoma Secondary to Swelling of the Lens.

PAUL STERNBERG AND SAMUEL J. MEYER (*American Journal of Ophthalmology*, May, 1950) are of the opinion that when an intumescent cataract produces acute glaucoma, then such an eye is a truly preglaucomatous eye in which the swollen lens acts as a natural provocative test. Gonioscopic examination of the opposite eye has shown that the angle of the anterior chamber is of the narrow type. Post-operative gonioscopic examination of the affected eye reveals peripheral anterior synechiæ. The authors consider that if the patient is examined within twenty-four hours of the attack, then a basal iridectomy may be performed, but that after twenty-four hours an iridencleisis should be performed. Removal of the lens by the intracapsular technique and with the use of corneo-scleral sutures is performed six weeks later. The authors consider that removal of the lens at the same time as the antiglaucoma operation may increase the risk of post-operative glaucoma, since additional peripheral anterior synechiæ may develop if, for any reason, there is delay in the reformation of the anterior chamber. Miotics should be used in the opposite eye, and in addition lens extraction in this eye should not be delayed if lenticular changes are sufficient to account for decrease in vision.

Hyaluronidase in Ocular Surgery and Therapy.

JAMES E. LEBENSOHN (*American Journal of Ophthalmology*, June, 1950) describes the properties and clinical value of hyaluronidase. He considers that hyaluronidase is the most important adjuvant to local anæsthesia since the introduction of epinephrine. To a five-millilitre vial containing five turbidity-reducing units of hyaluronidase was added an amount of one to three millilitres of procaine-epinephrine

solution. Lid œdema associated with the Van Lint method is transient and negligible, while in the O'Brien technique akinesia is equally effective. In either case the akinesia is so great and prolonged that lid suture is mandatory. In operation on the extra-ocular muscles, injection of the ciliary ganglion was quite sufficient. Enucleations were carried out without pain. In tear-sac surgery hyaluronidase produced an ischæmic field. The effects of retrobulbar injection of procaine and epinephrine on intraocular tension and pupillary dilatation are intensified by hyaluronidase, and accordingly it should not be used in operations for uncomplicated cataract or in glaucoma operations in which spontaneous prolapse of the iris is desired. The author recommends its use in chalazion operations, Ziegler cautery, electrolysis *et cetera*, where distension may disturb the procedure. He recommends its use also to reduce post-operative œdema after enucleations, and to reduce tension in glaucoma. He has found that subconjunctival injections of five turbidity-reducing units in 0·75 millilitre of procaine-epinephrine solution have reduced the hypopyon associated with iritis and corneal ulceration. In the treatment of corneal ulcer by subconjunctival penicillin injection he recommends that hyaluronidase be incorporated. In iritis hyaluronidase added to cocaine, atropine and epinephrine freed posterior synechiæ of one week's duration. In glaucoma due to iritis hyaluronidase injected subconjunctivally is worthy of trial before resort is had to paracentesis. Retrobulbar injections of hyaluronidase are recommended in thyreotoxic exophthalmos.

OTO-RHINO-LARYNGOLOGY.

Modified Radical Mastoid Operations with Meatal Preservation.

M. R. SHERIDAN (*The Journal of Laryngology and Otology*, May, 1950) discusses modified radical mastoid operations with meatal preservation. He states that the usual post-aural incision is made. The periosteum is raised with a narrow elevator, so that the periosteum and membranous meatus form a continuous sheet. The posterior and superior walls of the membranous meatus are divided level with the mastoid surface, with resultant avoidance of all tension on the meatus when the mastoid retractor is opened; otherwise there is a tendency for detachment to take place at or near the tympanic ring. The membranous meatus can now be elevated off the superior and posterior walls of the bony external meatus, but continuity of skin and tympanic membrane are carefully preserved. The mastoid bone is next excavated in the usual manner until antrum, aditus and attic are exposed. The bridge, incus and head of the malleus are all removed and granulations and cholesteatoma are cleared away. The tympanic membrane can be carefully lifted and the drum cavity inspected, and if necessary, cleaned. Penicillin powder is put into the cavity and the incision is completely sutured. Finally the meatus is completely filled with ribbon BIPP gauze. The severed surfaces of the membranous meatus come into contact and heal together. Stenosis does not occur. The dressings and sutures are removed after one week. The meatus is repacked for a further week, after which daily mopping out and powdering are carried out until the ear is dry. Sound healing is attributed to the clean attic, aditus and antrum, the complete normal meatal skin continuous with the tympanic membrane, and the small mastoid bone excavation. Of 33 cases forming the basis of the report, healing was complete in 29 and took variable times between two weeks and twenty-eight weeks. There was some temporary recurrence of discharge in four cases, and only one was regarded as a failure. Hearing was improved in 15 cases, unchanged in 10, worse but still serviceable in five, and not serviceable in only two cases; in one of the last-mentioned it was not serviceable before operation.

Rhinotomy for Exploration.

J. E. BORDLEY AND W. P. LONGMIRE (*Annals of Otology, Rhinology and Laryngology*, December, 1949) state that inadequate exposure is often a limiting factor in performing certain extensive surgical procedures in the nasal passages and accessory sinuses. The nose may be reflected to one side, remaining hinged by the soft tissues of that side. To achieve this an incision is made on one side through the facial attachment of the *ala nasi*, and thence upwards along the naso-maxillary fold to the level of the naso-frontal suture. The nasal bones are separated, together with the frontal process of the maxilla on each side, and the bony bridge is cut across along the line of the naso-frontal suture. The septum is cut across up to the level of the incision across the glabella. After bleeding points on the lateral nasal wall and on the septum have been controlled, a submucous resection of the remaining septal cartilage and perpendicular plate of the ethmoid bone may be performed, so that the septum may more readily be deflected to either side, giving an excellent exposure of the ethmoid and sphenoid sinuses. If exploration of the frontal sinuses is necessary, secondary incisions are made from the outer angles of the transverse glabellar incision up along the eyebrow. The whole of the anterior wall of the frontal sinus and its floor may be removed in order to obliterate the dead space in this sinus. The nose is replaced by first suturing the two portions of the septum together, and then closing the incisions across the glabella, the columella and the lateral naso-maxillary fold. Care is essential to approximate the bony edges of the maxillo-frontal process accurately, and further security is assured by the application of an external splint of "Stent" dental compound. The subsequent facial scarring is minimal. Septal perforation has not occurred. The operation is indicated in cases in which the standard types of intranasal operations would not provide adequate exposure.

Treatment of Juvenile Papilloma of the Larynx with Resin of Podophyllum.

J. B. HOLLINGSWORTH, H. W. KOHLMOOS AND R. C. MCNAUGHT (*Archives of Otolaryngology*, July, 1950) state that Sullivan in 1949 showed that podophyllum produced arrest of mitosis and other cytotoxic effects. Cellular destruction is more selective for young embryonic and tumour cells than for adult cells. The authors present reports of five cases of laryngeal papilloma in young children treated with local application of a 15% solution of podophyllum in 95% alcohol. All had had a great variety of previous therapy without improvement. The treatments were given sometimes daily at first, then at weekly intervals and then monthly. At each sitting some of the papillomatous tissue was removed. It was noted that the papillomata developed a grey tint immediately after the solution was applied, while normal tissue showed no change. The ease of subsequent removal of the lesions was remarkable as compared with the great difficulty previously encountered. Recurrences apparently were minimized. No untoward reactions were noticed.

The Incidence of Total Laryngectomy.

HAYES MARTIN (*Annals of Otology, Rhinology and Laryngology*, June, 1950) has made a survey to determine the frequency with which the operation of total laryngectomy is being performed in the United States of America and in other countries. American figures are more complete than those from other parts. The operation was performed in at least 846 cases in the United States during the year 1947. In France there were 115 cases, in Spain 122, in Argentina 91 and in Great Britain 35. There were records of only two cases from Australia. The data from various American cities suggest that the operation is most frequently performed in the larger cities where there are clinics specially organized and equipped for the purpose. The high incidence of the operation in some cities is due largely to the activities of a single clinic, sometimes even to the activities of a single surgeon.

House Dust Allergy.

T. G. RANDOLPH (*Archives of Otolaryngology*, July, 1950) states that the degree of sensitivity of an individual allergic subject is determined by performing intradermal skin tests with 0·01 millilitre of successive serial dilutions of the dust extract. The symptoms from house dust are more pronounced during the winter months when doors and windows are closed and fires are burning. The degree of sensitivity increases from mid-summer to mid-winter or late winter in many cases. During the winter of 1946-1947 the optimum effective dose of house dust extract was determined in a series of cases, the information serving as a guide for determining the current effective dose. This has been helpful in aiding one to reach an effective level of therapy in a minimum of time and with minimal reactions. A calculated maximum therapeutic dose is able to be approached in three doses given on alternate days or every third day. The therapeutic dose may be considered adequate if the injection is followed by partial relief of symptoms. The duration of the period of relief once an effective maintenance dosage level has been attained is remarkably constant for a given patient, so that he may be placed on a schedule of treatment a day or two short of the "wear off" time.

Special Abstract.

WERDNIG–HOFFMANN'S INFANTILE PROGRESSIVE MUSCULAR ATROPHY AND ITS RELATION TO AMYOTONIA CONGENITA.

THE primary object of the investigation made by Sven Brandt in Denmark between 1944 and 1948 was to determine from a critical examination of the literature and of clinical material collected from Danish clinics whether Oppenheim's *amyotonia congenita* was a well-defined and proven disease. The results of that investigation have just been published and are found to support the impression of neurologists interested in the problem that *amyotonia congenita* is merely a clinical syndrome and not a disease *per se*.

Generalized muscular hypotonia was the most important feature of the two diseases found to demand most attention in Brandt's study. Oppenheim's *amyotonia congenita* was originally named by him *myatonia congenita*, but later in 1908 was renamed *amyotonia congenita* on the suggestion of James Collier and Kinnier Wilson to avoid confusion with *myotonia congenita* or Thomsen's disease. According to Oppenheim, the hypotonia did not indicate a real paresis, the condition was usually stationary or improved, and it might even subside completely. Werdnig-Hoffmann's infantile progressive muscular atrophy, on the other hand, was characterized by widespread and progressive muscular pareses, usually ended fatally at an early age owing to increasing weakness of the respiratory muscles complicated by intercurrent respiratory infection, and in a few cases led to extensive muscular atrophy and death of the patient during the second or early in the third decade of life. Understanding of the true nature of Oppenheim's *amyotonia congenita* and recognition of the true cause of the generalized muscular hypotonia at as early an age as possible were therefore of major clinical importance.

An historical survey of Oppenheim's *amyotonia congenita*, Werdnig-Hoffmann's infantile progressive muscular atrophy and congenital muscular dystrophy with similar features reveals an interesting situation in regard to these disorders.

In 1900 Oppenheim published a paper of two pages in which he named the condition he described *myatonia congenita*. The parents of the children complained that they moved less than other children and that their limbs had been more or less flabby since birth. Oppenheim found muscular hypotonia of the trunk and extremities, weakened or abolished tendon reflexes, increased mobility in the joints on passive movement, and reduction of spontaneous mobility to such an extent that the patient seemed to be paralysed. He recognized various degrees of severity, presented the condition as a clinical contrast to Little's disease, and assumed that it was due to defective development of the musculature or of the peripheral motor neuron. He provided no anatomical confirmation. He noted in some cases an improvement in the condition in the course of time. He quoted no clinical case records, but gave brief abstracts of cases later in 1904 and 1912. Examination of these two case records and of two more presented from his clinic by Habermann showed the following. The case presented in 1904 was that of a child, nineteen months old, with a minor degree of muscular hypotonia, and that in 1912 was one of a child, twelve years old, who had shown some improvement but was seriously disabled. The first patient described by Habermann as suffering from *amyotonia congenita* subsequently died and was found at autopsy to have changes in the spinal cord which were undoubtedly those of infantile progressive muscular atrophy. The second case described by Habermann was clinically of the same type. Thus the clinical picture of *amyotonia congenita* was indefinite, uncertain and blurred from the beginning.

In contrast with the description of *amyotonia congenita*, the original accounts of infantile progressive muscular atrophy were thorough and comprehensive in their presentation of the clinical features and of the pathological changes associated with them. In 1891 and 1894 Werdnig described from Graz, Austria, two brothers who had suffered from progressive pareses from the first year of life and had died at the ages of three and six years. He found at autopsy extensive degenerative and atrophic changes in the motor cells of the spinal cord. He classified the condition as a transitional one between spinal and myogenic muscular atrophy with lesions in the spinal cord and dystrophic features; and pointed out that in 1888 Heubner had described a case of spinal muscular atrophy beginning at the age of three years and leading to death of the patient at the age of twenty-one years. Hoffmann, working in Erb's clinic at Heidelberg, described in 1893, again in 1897 and again in 1900, seven patients from four different families and identified his cases and those reported by Werdnig as examples of a hitherto unrecognized spinal muscular atrophy in children. Since then others have described about 200 cases from 120 families. In England Beevor described the first case of congenital, familial spinal muscular atrophy in 1902; and in 1911 Batten considered that the immediate cause of progressive muscular atrophy of the Werdnig-Hoffmann type was degeneration somewhere in the peripheral neuron, which need not necessarily involve the anterior horn cells, could be demonstrated in some cases only in the anterior spinal nerve roots, and might involve only the distal nervous structures or the peripheral parts of the nerve fibres.

In 1903 Batten applied the term "simple atrophic type of myopathy" to a stationary muscular disorder which presented itself as a congenital universal muscular hypotonia with reduction in muscular power and absence of tendon reflexes; and in 1911 considered Oppenheim's *amyotonia congenita* to be a benign congenital form of muscular dystrophy. In 1939 Aldren Turner the younger presented further observations on Batten's cases in adult life, showed that the original universal hypotonia without local muscular atrophy had been replaced by local atrophy of the proximal muscles of the limbs with disappearance of the general hypotonia, and confirmed Batten's assumption that their condition was an unusually benign type of muscular dystrophy. Finally in 1949 he published the findings at autopsy in one of Batten's cases and confirmed the diagnosis of muscular dystrophy.

Thus from a critical review of the literature on the conditions associated with muscular hypotonia in infancy and early childhood, the position appears to be as follows. Werdnig-Hoffmann's disease is a well-defined condition about which there has been no doubt since it was first described fifty years ago. Later it was found that it might be congenital, that it might take for a time a stationary course and even seem to improve, and that it might spread in its later stages to involve the cranial nerve region. It may be inherited recessively, though that is uncertain because of the small number of cases that have been available for study. It sometimes appears in a transitional form between the classical condition and other abiotrophic muscular and nervous disorders. It is most often rapid in its course, but it sometimes develops more slowly, and later appears in a juvenile form with more diffuse extensions in the central nervous system. Muscular dystrophy with a clinical course similar to that of the classical Werdnig-Hoffmann's atrophy has been assumed to be present in a small number of cases in which post-mortem examination shows the nervous system to be undamaged. Oppenheim's *amyotonia congenita* was a less clearly defined condition even when it was first described; it often resembled Werdnig-Hoffmann's disease clinically and it was presumably confused with it by Oppenheim himself. Most of the cases that have been reported as *amyotonia congenita* are found on critical analysis to be cases of Werdnig-Hoffmann's disease. The remaining cases described as *amyotonia congenita* follow such a benign course that they seem to occupy a special position. In spite of the features they have in common they differ so much in other features that it is impossible to regard them as examples of one and the same disease. Rather they are a number of morbid conditions which may be grouped clinically as Oppenheim's *amyotonia congenita*. Some cases in which that syndrome is present may be examples of a slowly, almost unnoticeably, progressive atrophic muscular affection, characterized in infants by universal hypotonia, and of the nature of the condition described by Batten and more recently by J. Aldren Turner as a congenital form of muscular dystrophy. Finally it is possible that there are a number of relatively mild cases of congenital flabbiness of muscles and joints, of the type described by Catel, Sobel and Zellweger, so seldom studied that their nature and possible pathological uniformity have not been recognized.

Upon that background Brandt analyses his own material. He collected 156 cases, 131 of them primary cases and 25 secondary cases. He classified as primary, cases in older persons found recorded in Danish clinics and new cases diagnosed by himself; and as secondary cases those observed in siblings of patients in the primary case group.

[1] "Werdnig-Hoffmann's Infantile Progressive Muscular Atrophy: Clinical Aspects, Pathology, Heredity and Relation to Oppenheim's Amyotonia Congenita and other Morbid Conditions with Laxity of Joints or Muscles in Infants", by Sven Brandt; 1950. Copenhagen: Ejnar Munksgaard. 6¼" × 9½", pp. 360, with illustrations. Price: Danish Kr. 20.

Of the 131 primary cases, he carried out examination in 32 of the old cases and in 28 of the new cases, a total of 60; and supplemented the clinical records of the other cases by information from the parents of the children. Of the 25 secondary cases, he carried out personal examination in three, examined the case records of seven upon which various diagnoses had been made in Danish clinics, and obtained an account of the remaining 15 patients from the parents.

Among the 71 cases in which the diagnosis had been *amyotonia congenita*, he diagnosed 37 patients as suffering from Werdnig-Hoffmann's infantile progressive muscular atrophy, one as suffering from another degenerative condition of the nervous system, and 12 as suffering from other conditions. He retained the diagnosis of *amyotonia congenita* in only 10 cases.

Among the total of 131 primary cases originally diagnosed as *amyotonia congenita*, spinal muscular atrophy (probably congenital), *amyotonia congenita* (Werdnig-Hoffmann), progressive muscular dystrophy and similar conditions, he diagnosed 112 patients in all as suffering from Werdnig-Hoffmann's infantile progressive muscular atrophy and only 13 as suffering from *amyotonia congenita*.

Having so grouped the available case material, he presents a detailed study of the 112 cases of infantile progressive muscular atrophy. He defines that condition as follows: "A heredofamilial, progressive lesion in the peripheral motor neuron or in the musculature, commencing in fetal life or within the first two years of life, bringing about extensive, as a rule symmetrical, flaccid pareses, muscular atrophy, loss of tendon reflexes, and, if death does not appear within a few months, gradually increasing contractures. Most of the patients die within the first year of life, but almost all die before the age of 20, in a state of complete morbidity."

He bases his account of the clinical features of the disease on the revised data from 91 cases, in 31 of which the patients were observed or reexamined by himself. Considering first the symptoms in 45 infants, for it was in them that the clinical picture was often called in error *amyotonia congenita*, he regards as the most important the posture of the child, the type of respiration, hypokinesia, loss of muscular power, hypotonia, hyperflexibility, primary contractures, areflexia, muscular atrophy and skin changes. The child is usually motionless except for movements of the eyeballs and of the diaphragm. He is in a position of flexion of the upper limbs with pronation of the forearms and of slight flexion and extreme outward rotation of the lower limbs to bring them into a "frog-like" posture. Respiration is accelerated and of the reversed type. The subcutaneous adipose tissue is well developed and variously described as pasty, thickened, fibrous or infiltrated in a peculiar way. After the first year of life, the hypokinesia, hypotonia and reduction in muscle power are less conspicuous; the muscular atrophy is easier to demonstrate; muscular fibrillation is seen more often; contractures are more common and hyperflexibility is less.

Electromyography in 19 cases gave less reliable results in the first months of life than histological examination of the muscles on biopsy; but synchronism in two of three leads from the same muscle supported a diagnosis of spinal muscular atrophy. The excretion of creatine was increased in the eight cases in which it was investigated. Histological examination of excised muscle tissue gives information on the presence of atrophy, and, when it is present, on the nature of the atrophy, whether spinal or myogenic in origin; this examination was made in 23 cases. In 12 cases it supported the diagnosis of spinal muscular atrophy, which was verified at autopsy in four cases; in 11 cases it failed to reveal a spinal type of atrophy, though in one case autopsy two months later revealed typical changes in the muscles and lesions in the spinal cord; and in four cases it showed such severe, widespread atrophic changes that no conclusion could be reached on the origin of the atrophy. It gave its most reliable results in infants; it was sometimes inconclusive in older children with more advanced atrophic changes, but it might still be useful in them if care was taken to select only muscles with moderate atrophy for biopsy.

Hoffmann regarded infantile progressive muscular atrophy as a condition of early onset, but not necessarily congenital. Beevor and others, however, described cases which were congenital. Brandt stated that in 12 cases of his series there was a history of feeble or diminishing fœtal movements, and that in 41 cases the condition was present at birth. The onset was before the age of six months in 73 cases and before that of twelve months in another 24 cases; that is, before twelve months in 97 of the 112 cases. It was after the age of twelve months in nine cases, and at an unknown age in six. The course of the disease was usually rapid with early death, but in some cases it was rather slower with stationary periods. Of the 112 patients, 95 had died by the time the investigation was completed, one-half of them before the age of twelve months and four-fifths before the age of four years. The oldest patient in the series was twenty years old and completely disabled.

The pathological anatomy of the condition described by Brandt is based on the findings at 16 autopsies and 23 muscle biopsies. The characteristic change in the muscles was a patchy atrophy, which was found in association with atrophy of the anterior spinal roots and degeneration of the large cells of the anterior horns of the spinal cord and in a lesser degree of the motor nuclei of the cranial nerves. It was found, however, in a rare case in which the anterior horn cells of the spinal cord were histologically normal and there were no other changes. It was most distinct in infants, in whom histological examination was most often needed to establish the diagnosis, and easy to demonstrate by muscle biopsy. It tended to become obscured in children over the age of one year with a slower clinical course, as the atrophy became more extensive, the supporting connective tissue proliferated and became infiltrated with fat cells, and the histological picture came gradually to resemble that of advanced muscular atrophy in adults. Sometimes a more diffuse and non-patchy form of atrophy was found on biopsy and was interpreted as indicating a condition of a myogenic nature. That variation could not be so interpreted, however, in children over the age of one year, for in them it resembled closely the more extensive atrophy of cases of infantile progressive muscular atrophy with a slower clinical course.

Hoffmann suggested that infantile progressive muscular atrophy was due to an inherited lack of vitality of the peripheral motor neuron, and that it belonged to the group of abiotrophic diseases which had in common the precocious death of an organ system or of certain parts of it. Brandt regards the disease as due primarily to a recessively transmitted gene, possibly at times to an incompletely dominant gene, which leads to poor utilization of certain food elements essential to the metabolism of the particular cells. He found no relationship between the condition and diseases suffered by the mother during pregnancy, complicating birth injuries, the diet of the mother during pregnancy, the diet of the child or infections during the early months of life. The primary site of the disease was the peripheral motor neuron and the muscular atrophy was secondary to the atrophy of the nerve cells. In rare cases, however, the pathogenic factors appeared to act primarily on the muscle cell and to cause no apparent changes in the peripheral motor neuron.

Treatment of the condition was restricted to palliative and prophylactic measures. No patient proven to have the disease was known to have had offspring. In regard to whether it was justifiable to take steps to discourage or to prevent established conductors of the disease from having children, it was decided that it was very doubtful whether in such cases interruption of pregnancy could be advised for eugenic reasons alone.

In the examination of the differential diagnosis of Werdnig-Hoffmann's disease Brandt provides a useful survey of the other morbid conditions of infancy characterized by laxity of muscles. From the literature and his own material he describes many other conditions which have been confused with infantile progressive muscular atrophy of the Werdnig-Hoffmann type. Among them are atypical forms of progressive muscular atrophy, amaurotic familial idiocy, diffuse sclerosis of the brain in cases in which the muscles are flabby, hereditary amyotrophic lateral sclerosis of the type described by Ford, and hereditary ataxia of the Friedreich type. Conditions based upon various forms of agenesia in the nervous system are congenital cerebellar ataxia, atonic congenital diplegia, initially hypotonic forms of spastic diplegia of the type of Little's disease, and symptomatic cerebellar ataxia and diplegia with initial hypotonia from natal intracranial hæmorrhage in the cerebellum and brain stem. Other conditions less often confused are birth injury to the cervical portion of the spinal cord and spinal nerve roots, idiocy, imbecility with muscular hypoplasia and hypotonia, hydrocephalus with muscular flabbiness, cerebral atrophy with internal hydrocephalus secondary to encephalitis, radiculoneuritis, polymyositis, tumour of the fourth ventricle, and universal muscular hypoplasia. Conditions rarely confused are rhachitic myopathy and muscular flabbiness associated with protracted dyspeptic conditions, cœliac disease or Addison's disease. Congenital myasthenia is a possible source of confusion, but, because of the pronounced bulbar symptoms usually present, is unlikely to cause serious difficulty.

Brandt retained the diagnosis of *amyotonia congenita* in 13 cases only. He points out that he uses it merely as a symptomatic diagnosis, and considers that among his cases there must have been several conditions, but he has found no definite answer as to their nature. He is tempted to apply to some of them the term suggested by Batten—congenital, non-progressive, muscular dystrophy—but, in the absence of anatomical proof, refrains from doing so.

British Medical Association News.

SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held at Ballarat on Saturday, November 25, 1950, DR. ROBERT SOUTHBY, the President, in the chair. The meeting in the afternoon took the form of a series of clinical demonstrations by members of the honorary medical and surgical staffs of the Ballarat Base Hospital. Parts of this report appeared in the issues of February 17 and 24, 1951.

Chronic Osteomyelitis with Malignant Changes.

DR. M. ROBINSON showed a patient with an epithelioma arising in an osteomyelitic sinus. The patient, a man, aged fifty-one years, had developed about eighteen months previously typical acute osteomyelitis of the left femur, which necessitated operation and evacuation of pus. The post-operative treatment included administration of sulphadiazine, penicillin and streptomycin, and the patient responded after some days. On discharge from hospital he was afebrile, and there was still a slight discharge from his sinus. Four months later he was admitted for sequestrectomy under penicillin and streptomycin cover. That was uneventful, but the sinus did not close, and some three months later a second sequestrum was seen on X-ray examination; sequestrectomy was again performed with extensive removal of overhanging bone. He was discharged from hospital well, and walking on crutches. However, the sinus persisted, although the discharge was sterile on attempted culture, and some seven months before the meeting a further operation was carried out; the old scar tissue was excised and sequestra and granulations were removed. Primary union was not obtained, but the patient left hospital afebrile and the wound was sterile. However, he reported that on several occasions there was quite brisk and extensive hæmorrhage from the sinus, and further X-ray examination showed bone destruction and invasion extending in the shaft of the femur. A diagnosis of malignant disease was made. No evidence of metastases could be found. Operation was therefore performed, three months before the meeting, and amputation through the upper third of the thigh was carried out. The specimen was shown microscopically to be a squamous-celled carcinoma.

Urological Cases.

DR. G. R. DAVIDSON discussed four cases of urological interest.

The first was that of a calculus removed from the ureter by means of a looped catheter.

The second was that of a large hydronephrosis removed from a man whose opposite kidney showed radiological evidence of some scattered calcification, but otherwise appeared to be healthy.

The third was that of a testicle removed from the left inguinal canal of a youth of eighteen years. The testicle appeared normal on inspection, but biopsy performed at the time of operation failed to reveal any evidence of function.

The last was that of a girl, aged eighteen years, who had suffered from urinary incontinence since birth; the incontinence occurred in association with congenital absence of the sacrum. It had been relieved by the operation of pre-sacral neurectomy. Pre-operative and post-operative treatment was discussed at some length.

Abdominal Mass for Diagnosis.

DR. W. R. GRIFFITHS showed a woman, aged sixty-nine years, who had had recurrent attacks of colicky pain in the right hypochondrium for fifteen years, since removal of her gall-bladder. Her common bile duct had been explored eight years before the time of the meeting, and after that operation she was free from symptoms for twelve months; then the pain recurred. With the more severe attacks of pain, vomiting was present. She was afraid to eat any food, particularly fatty foods, as they were likely to increase the pain. Examination could detect nothing abnormal except some rigidity around the scar in the right hypochondrium and tenderness in the epigastrium. Investigations were carried out, and urine examination showed a faint trace of bile, but no urobilin. The Van den Bergh test yielded a positive direct reaction, the serum bilirubin content was 3·1 milligrammes *per centum*, the alkaline phosphate level was 74 units and the result of the cephalin flocculation test was negative. The findings from blood examination were otherwise normal. X-ray examination showed a rounded shadow about half an inch in diameter opposite the transverse process of the second lumbar vertebra. Dr. Griffiths presented the patient for diagnosis.

Hydatid Disease of the Liver.

Dr. Griffith's second patient was a man, aged fifty-two years, with a previous history of an operation for hydatid cyst of the liver some forty years before. On the present occasion he had complained of epigastric fullness with intermittent twinges of pain in the same area. Examination showed a cystic swelling in the epigastrium; the swelling was tense and a little tender. X-ray examination confirmed the presence of a tumour in the area and showed the right hemidiaphragm to be raised three inches. Operation was carried out some five months before the time of the meeting, and a hydatid cyst in the abdominal wall was found to communicate with a large cyst in the left lobe of the liver. That in turn was found to communicate with a still larger cyst lying underneath the diaphragm. The cysts were all evacuated and part of the wall of the cyst in the liver was removed; the opening of the cavity was closed around a drainage tube. Severe secondary hæmorrhage occurred which necessitated blood transfusion, but that finally ceased and free biliary drainage occurred; more recently the drainage had stopped, and the patient was well and had gained two stone in weight.

Arachnodactyly, Abdominal Pregnancy and Albright's Syndrome.

DR. M. ROBINSON demonstrated a case of arachnodactyly and DR. T. GREENING demonstrated cases of abdominal pregnancy and Albright's syndrome.

Intravenous Procaine Administration.

DR. W. A. PRYOR and DR. J. C. DICK read a paper entitled "Intravenous Procaine".

Correspondence.

THE HOSPITAL ALMONER PROBLEM.

SIR: Members of the Australian Association of Almoners welcomed your editorial of January 20, 1951, in which the work of almoners was outlined, the shortage stressed, and the need emphasized for higher salaries and status if suitable people are to be encouraged to enter the field.

In its twenty-one years of existence in Australia the profession of medical social work has developed considerably in its training and practice. Almoners in Australia have not undertaken administrative and financial responsibilities such as assessment of patients' payments which devolved earlier upon almoners in Great Britain. They have been free, therefore, to devote themselves to the study and relief of patients' social, economic or emotional needs related to illness and injury as cause or effect.

The growing concern of the medical profession with the social and psychological factors in medicine has produced an increasing demand for the service of almoners to work closely with doctors in helping patients to achieve through their own and community resources the fullest possible restoration to health and satisfying useful activity. This service includes practical arrangements for financial help, transport, convalescent or institutional care, suitable training, employment and recreation. It includes helping the patient and his family through skilled interviewing to relieve anxiety, to achieve healthy attitudes towards disability and treatment, and to modify disturbed social relationships as they contribute to ill-health. Almoners are asked increasingly to assist with teaching the social component in medicine to medical, nursing and other students, to share in medical social research, to participate in com-

munity activities related to the medical social needs of individuals and the community as a whole.

There are at present many established positions for almoners unfilled in Australia. In addition there are wide fields such as work with tuberculous patients, with maternal and infant welfare, which are comparatively untouched and where development only awaits the promise of staff.

Several factors militate against the flow of recruits. Salaries are low in comparison with those paid in many other professions and in other fields of social work. Existing almoner departments are with few exceptions grossly understaffed. Excessive pressure of work makes it difficult to improve and develop standards of work. Clerical assistance is all too often inadequate, so that the almoners are burdened with inappropriate routine tasks. The offices allocated to almoners often lack the privacy and convenience which are essential for the nature of the work which is highly confidential to the patient. The status accorded to medical social workers in some hospitals and government services is inadequate for the responsible, skilled and arduous work which they are called upon to perform.

It is true that we do not seek as members of our profession people who are not imbued with a true spirit of service and deep concern for those in trouble. It is equally true that congenial conditions and adequate reward make possible the translation of this spirit into effective practice.

The generous guidance and support of many members of the medical profession have enabled medical social work to reach its present stage. Your editorial is gratifying evidence that we may hope for continued help in attaining the highest standards of work and the members required to maintain them.

Yours, etc.,

(MISS) ALISON PLAYER,
President, Australian Association of Almoners.

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Alfred Hospital,
Commercial Road,
Prahran, S.1,
Victoria.
February 12, 1951.

SIR: Permit me to compliment you upon the editorial in your issue of January 20, 1951, relating to the hospital almoner problem.

I endorse your comments, which stress the serious situation of hospitals because of the shortage of trained hospital almoners. If hospitals are to assist in the development of a positive preventive health programme, they must have almoners in adequate numbers, and properly trained. Further, it is apparent that proper use of hospital beds depends upon domiciliary medical care. This cannot be implemented without almoners.

In your list of a hospital almoner's duties, you mention the collection of hospital patients' contributions. In this hospital we have freed the almoner from the onerous task of collecting money, which formerly occupied a good deal of her time. We include in her duties the assessment only of an indigent patient's ability to pay. I believe this is the proper attitude to adopt if we are to save valuable almoner time.

Yours, etc.,

JOHN LINDELL,
Medical Superintendent.

The Royal Melbourne Hospital,
Grattan Street,
Parkville, N.2,
Victoria.
February 15, 1951.

SIR: I was gratified to read your leading article of January 20, 1951, discussing the problem of the hospital almoner. I wish to support wholeheartedly your commendation of their work. Surely the advancing front of medicine must enter deeply into the problems of social medicine. In Melbourne, and certainly elsewhere in Australia, the hospital almoners have contributed greatly to our appreciation of the forces which contribute to the ill-health of the community as well as giving great comfort to the sick.

In our unit at The Royal Melbourne Hospital we have been most fortunate in having an almoner. She has given aid and guidance to very many of our patients and has added much to our knowledge of disease.

It is the duty and privilege of the medical profession to extend a helping hand to the almoner service by making maximum use of their services and by giving every encouragement to those young women of Australia who may wish to enter upon a career which will render sterling service to mankind.

Yours, etc.,

IAN WOOD.

The Royal Melbourne Hospital,
Grattan Street,
Parkville, N.2,
Victoria.
February 9, 1951.

ACUTE ANTERIOR POLIOMYELITIS.

SIR: I think that I know as much or as little about poliomyelitis as my fellow graduates. The relationship between varicella and *herpes zoster* was long disputed, but now it is admitted that such is the case. It is interesting to note that an epidemic of varicella in Sydney was followed by a great increase in the number of cases of poliomyelitis. It would be interesting to know if there was an increase in the number of cases of *herpes zoster*. In my small way when poliomyelitis was about here and since I have seen an unusual number of cases of "shingles".

The pathological histology of shingles is astonishing alike to the changes in poliomyelitis, that is, in the degenerative changes found in the post-ganglia in *herpes zoster* and in the cells of the anterior cornual cells in poliomyelitis. I am writing this to suggest the use of "Contramine" in poliomyelitis. This preparation is almost a specific in the treatment of herpes.

Yours, etc.,

J. FORBES MACKENZIE.

Melbourne,
Undated.

THE SYNDROME OF LAMELLAR CEREBELLAR DEGENERATION ASSOCIATED WITH RETINITIS PIGMENTOSA, HETEROTOPIAS, AND MENTAL DEFICIENCY, WITH REPORT OF A CASE.

SIR: The histological findings given as evidence of the presence of *retinitis pigmentosa* in a case of lamellar cerebellar degeneration reported by P. H. Hagen, K. B. Noad and Oliver Latham (THE MEDICAL JOURNAL OF AUSTRALIA, February 10, 1951) are not convincing. There are many pathological conditions of the retina which lead to degeneration of the three layers of neurons with migration into the retina of retinal degeneration. The severe shrinkage associated with paraffin embedding adds to the difficulty of diagnosis. The only opinion that can be given from the evidence presented is that retinal degeneration is present. There is nothing to indicate its cause.

It is further stated that the ganglion cells are destroyed. If this is so, optic atrophy must be present, and this would be clearly demonstrated by a cross-section of the optic nerve prepared by the method of Weigert-Pal.

On the evidence presented there appears to be no justification for labelling this case as an example of the "syndrome of lamellar cerebral degeneration, associated with retinitis pigmentosa, heterotopias and mental deficiency".

Yours, etc.,

KEVIN O'DAY.

33 Collins Street,
Melbourne, C.1,
February 15, 1951.

PNEUMONIA IN POLIOMYELITIS.

SIR: It was of interest to learn that pneumonia was the end of a number of the recent poliomyelitis victims.

At the moment I am assisting in the care of a young man of twenty-three, whose behaviour has illuminated this statement. He has extensive pareses involving lower limb, back, abdominal and intercostal muscles. His diaphragm functions normally. When asked to cough he utters an attenuated grunt. In fact he cannot cough, and therein lies his danger.

The following sequence took place on successive days. First day: He suddenly became unconscious and black in the face, but returned to normal upon intubation and removal of the sputum from his trachea. Second day: he achieved a complete clinical and radiographic collapse of his right lung, which was relieved at once by bronchoscopy.

Third day: he repeated his performance of the first day. Following these incidents he was intubated six-hourly and his bronchi sucked out. Four days later, for convenience sake, a tracheotomy was done.

One hazards the assumption that not a few of the pneumonias are really cases of sputum retention and pulmonary collapse. Early, adequate and repeated removal of secretion will alone prevent true pneumonia, and a possibly fatal termination.

Yours, etc.,

189 Macquarie Street, Sydney,
February 13, 1951.

HARRY M. WINDSOR.

THE COMMONWEALTH JUBILEE NUMBER.

SIR: In the Commonwealth Jubilee Number of THE MEDICAL JOURNAL OF AUSTRALIA under the heading of "Dermatology", it was stated that the Dermatological Association of Australia was publishing a yearly volume of transactions. As this is not quite correct, I would be grateful if you would either publish this letter or make an announcement to the effect that the Dermatological Association of Australia is publishing a journal to be known as *The Dermatological Journal of Australia*. It will be published every six months for a start and will contain scientific papers from the members and honorary members, and is open to dermatologists of other countries for the publication of papers. Transactions may also be included.

Yours, etc.,

J. C. BELISARIO,
Lecturer in Dermatology to the University of Sydney.

143 Macquarie Street, Sydney,
February 13, 1951.

Public Health.

DIAGNOSTIC CONSULTANTS IN NEW SOUTH WALES.

AT the request of the Secretary of the Hospitals Commission of New South Wales, the following list is republished of physicians in Sydney and Newcastle who are prepared to act as diagnostic consultants in regard to poliomyelitis. The list was previously published in the issue of December 10, 1949.

The Royal Prince Alfred Hospital:

Dr. C. G. McDonald, 143 Macquarie Street, Sydney. Telephones: BU 2071, FU 7982.
Dr. W. P. MacCallum, 143 Macquarie Street, Sydney. Telephones: BU 1149, FM 4266.

Saint Vincent's Hospital, Darlinghurst:

Dr. Bruce Hall, 185 Macquarie Street, Sydney. Telephones: BW 9044, XB 5013.
Dr. P. J. Markell, 193 Macquarie Street, Sydney. Telephone: BW 7126.

Sydney Hospital:

Dr. A. Hogan, 139 Macquarie Street, Sydney. Telephones: BW 4541, WL 1337.
Dr. F. Ritchie, 225 Macquarie Street, Sydney. Telephone: BW 9071.

Royal Alexandra Hospital for Children:

Dr. S. G. Bradfield, 225 Macquarie Street, Sydney, Telephones: BW 9320, UY 1083.
Dr. Gertrude S. Geikie, 149 Macquarie Street, Sydney. Telephones: BW 8616, JX 2109.

The Royal North Shore Hospital of Sydney:

Dr. I. A. Brodziak, 231 Macquarie Street, Sydney. Telephones: BW 9910, FU 2005.
Dr. Douglas Anderson, 185 Macquarie Street, Sydney. Telephones: BW 6944, XF 1193.

The Royal Newcastle Hospital:

Dr. C. A. Clark, 20 Bolton Street, Newcastle.
Dr. A. T. Roberts, over 5 Union Street, Newcastle.
Dr. Ethel Byrne, Royal Newcastle Hospital.

Obituary.

EDGAR HENRY WARD.

WE are indebted to Dr. C. H. Dickson for the following account of the late Mr. Edgar Henry Ward.

Mr. Edgar Henry Ward, who had been Financial Secretary of the Victorian Branch of the British Medical Association since 1934, died on December 9, 1950, after many months of painful illness, which he bore with great fortitude. In his youth Mr. Ward joined the Tasmanian Government Railways, transferring to the Public Service of that State in 1915, when he became Secretary to the Premier, and later Secretary to the Hydro-Electric Committee. On retirement from the civil service, he founded a shipping firm in Hobart and represented the Commonwealth line of steamers, but when that line was sold he left Tasmania and, coming to Victoria, joined the staff of the Victorian Branch of the British Medical Association as Secretary to the Standing Insurance Committee, which was formed at that time to investigate schemes of national insurance. In 1934 he became Financial Secretary and remained in that office until his death, but during the war years served as Secretary to the Central Medical Coordination Committee in the Department of Defence.

An old friend of Mr. Ward's writes: We were friends for well over forty years and as boys swam, rowed and boxed together. I am therefore in a position to appreciate his worth as a man of sterling character. His death has robbed me of a very great and sincere friend. As a boy, he showed that he was a born leader, quick to sum up and take the initiative. He scorned liars, tricksters and deceitful people; hated cruelty, whether to humans or animals; and would fight fearlessly for his rights and those of others less able to do it for themselves. He was considerate and kind, and spent a lot of his time doing good turns for the sick and distressed.

At its meeting on December 13, 1950, the Council of the Victorian Branch of the British Medical Association recorded the following special minute:

> The Council of the Victorian Branch of the British Medical Association records with regret the death of Mr. Edgar H. Ward, who for twenty years, first as Secretary of the Standing Insurance Committee and, from 1934, as Financial Secretary of the Branch, rendered loyal and devoted service to the Association. Council extends its sympathy to Mrs. Ward and her son.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Clinical Meetings at Balmoral Naval Hospital.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that clinical meetings will be held at the Balmoral Naval Hospital, Balmoral, Sydney, during March and April, 1951, when the following lectures will be given:

Tuesday, March 13: "Medical Impressions of a Recent Trip Abroad", Dr. V. M. Coppleson.

Tuesday, April 17: "Some Aspects of 'Functional' or Psychosomatic Diseases", Dr. T. M. Greenaway.

Both these lectures will begin at 2 p.m., and after afternoon tea clinical cases will be shown by the hospital staff. All medical practitioners are invited to attend.

Course for Diploma in Dermatological Medicine.

Provided a minimum number of five candidates is offering, the next course for the diploma in dermatological medicine, Parts I and II, will begin in Sydney on June 18, 1951, and continue until March, 1952, prior to examinations. Intending candidates are asked to note that the closing date for applications is May 4, 1951, and early application is essential. Full details concerning examination requirements and courses for this diploma may be obtained on application to the Course Secretary, the Post-Graduate Committee in Medicine, 131 Macquarie Street, Sydney. Telephones: BU 5238, BW 7483. Telegraphic address: "Postgrad, Sydney."

Congresses.

THE OXFORD CONFERENCE ON TUBERCULOSIS AND DISEASES OF THE CHEST.

THE Oxford Conference (1951) on Tuberculosis and Diseases of the Chest will be held from July 17 to 21, 1951, as part of the Festival of Britain. Members may if they wish stay in College. Three days will be devoted to sessions of lectures and discussions at the Taylor Institution; subsequently members may if they so desire attend practical demonstrations that have been arranged at many chest centres. There will be a number of social events.

The cost of the conference will be £7 7s., which will include all meals, accommodation in College and the social events, including the dinner to be held on July 19. Those wishing only to attend the sessions of the conference will pay a fee of £1 1s. The honorary secretary of the conference is Dr. Stephen Hall, Bierton House, Bierton, Bucks.

TENTH ITALIAN TUBERCULOSIS CONGRESS.

THE tenth Italian Tuberculosis Congress will be held at the Instituto Sanatoriale "Principi di Piemonte", Naples, from Aprl 2 to 8, 1951. The subjects to be discussed will be Monaldi's drainage of cavities and B.C.G. vaccination. At the same time an international exhibition and conference of the medical Press will take place. The institute in Naples contains 2000 beds and cares for patients with all forms of tuberculosis. It is under the direction of Professor Vincenzo Monaldi. Visitors from Australia will be welcome and should get in touch with the secretary-general, Professor Gino Babolini, Instituto Sanatoriale "Principi di Piemonte", Napoli, Italy.

Australian Medical Board Proceedings.

NEW SOUTH WALES.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Practitioners Act*, 1938-1945, of New South Wales, as duly qualified medical practitioners:

De Bruin, Arthur James, M.B., B.S., 1945 (Univ. Ceylon), c.o. Dr. A. L. Caselberg, Corrimal.

Isles, James Llewellyn, M.B., B.S., 1949 (Univ. Queensland), 9 Thompson Street, Mosman.

Widdup, Alec Harley, M.B., B.Chir., 1941 (Univ. Cambridge), 115 Crebert Street, Mayfield.

Rail, Wilton Whitburn, M.B., B.S., 1941 (Univ. Melbourne), Sheridan Street, Gundagai.

Smith, George Birch, M.R.C.S. (England), L.R.C.P. (London), 1944, c.o. Dr. D. D. Gibson, Mullumbimby.

QUEENSLAND.

THE undermentioned have been registered, pursuant to the provisions of *The Medical Acts*, 1939-1948, of Queensland, as duly qualified medical practitioners:

Morton, Marian Beatrice, M.B., B.S., 1948 (Univ. Sydney), c.o. Flying Doctor Base, Cloncurry.

Statham, Cecily Faith, M.B., B.S., 1946 (Univ. Melbourne), D.O., R.C.O.G. (London), 1950, c.o. Dr. J. Stobo, Ballow Chambers, Wickham Terrace, Brisbane.

Grant, Frank John, M.B., B.S., 1950 (Univ. Melbourne), Mater Misericordiæ Public Hospital, South Brisbane.

O'Loughlin, Michael, M.B., B.S., 1950 (Univ. Melbourne), c.o. Mater Misericordiæ Hospital, South Brisbane.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED FEBRUARY 3, 1951.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.[2]	Australian Capital Territory.	Australia.[3]
Ankylostomiasis	*	..	..	..	..	..	..	..	..
Anthrax	*	..	..	..	..	..	..	..	..
Beriberi	*	*	*	*	*	*	..	*	..
Bilharziasis	*	..	..	..	..	..	..	..	..
Cerebro-spinal Meningitis	2(1)	2(1)	..	..	..	..	..	..	..
Cholera	..	..	..	..	..	..	..	..	..
Coastal Fever(*a*)	*	*	..	*	*	*	*	*	..
Dengue	*	*	*	*	*	*	..	*	..
Diarrhœa (Infantile)	..	..	2(1)	..	..	..	..	..	2
Diphtheria	11(2)	6(5)	5(4)	..	4(4)	1	..	..	27
Dysentery (Amœbic)	*	..	..	..	..	..	..	..	..
Dysentery (Bacillary)	*	2(2)	..	..	..	..	..	..	2
Encephalitis Lethargica	..	..	..	..	..	..	..	..	..
Erysipelas	*	*	*	..	*	*	..	..	..
Filariasis	*	..	..	..	..	..	..	..	..
Helminthiasis	*	..	*	*	*	*	*	*	..
Hydatid	*	..	*	*	*	*	*	..	..
Influenza	*	*	*	..	*	*	..	*	..
Lead Poisoning	*	*	..	*	*	*	*	*	..
Leprosy	..	..	..	..	..	..	..	..	..
Malaria(*b*)	*	..	..	..	2	..	..	..	2
Measles	*	*	*	38(8)	*	*	..	..	38
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	74(30)	10(3)	37(1)	22(13)	..	18(4)	..	..	161
Psittacosis	*	..	*	..	*	*	*	*	..
Puerperal Fever	..	..	2	1(1)	..	..	..	..	3
Rubella(*c*)	*	*	*	*	..	*	..	1	1
Scarlet Fever	13(6)	16(5)	10(5)	3(2)	2(2)	1(1)	..	..	45
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	*	..	..	*	*	*	*	*	..
Trachoma	*	..	*	*	*	*	*	*	..
Tuberculosis(*d*)	4(3)	17(11)	9(4)	5(4)	16(13)	7(1)	..	..	58
Typhoid Fever(*e*)	..	..	..	..	..	..	..	..	..
Typhus (Endemic)(*f*)	2	..	2	..	..	..	..	..	4
Undulant Fever	..	2	..	..	..	..	..	..	2
Weil's Disease(*g*)	*	*	8	*	*	*	*	*	8
Whooping Cough	*	*	*	2(1)	*	*	..	..	2
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] The form of this table is taken from the *Official Year Book of the Commonwealth of Australia*, Number 37, 1946–1947. Figures in parentheses are those for the metropolitan area.
[2] Figures not available.
[3] Figures incomplete owing to absence of returns from the Northern Territory.
* Not notifiable.
(*a*) Includes Mossman and Sarina fevers. (*b*) Mainly relapses among servicemen infected overseas. (*c*) Notifiable disease in Queensland in females aged over fourteen years. (*d*) Includes all forms. (*e*) Includes enteric fever, paratyphoid fevers and other Salmonella infections. (*f*) Includes scrub, murine and tick typhus. (*g*) Includes leptospiroses, Weil's and para-Weil's disease.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Caspersonn, Douglas James, M.B., B.S., 1951 (Univ. Sydney), Balmain and District Hospital, Balmain.

Mulhearn, Richard John, M.B., B.S., 1951 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Sonnabend, Samuel Isaac, registered in accordance with the provisions of Section 17 (1) (c) of the *Medical Practitioners Act*, 1938-1945, 57 Hawkesbury Road, Westmead.

Channells, Raymond Robert, M.B., B.S., 1951 (Univ. Sydney), Royal North Shore Hospital, St. Leonards.

Walsh, Clement Henry, M.B., B.S., 1944 (Univ. Sydney), 7 Victoria Avenue, Middle Cove, East Willoughby.

Keller, Arthur William, M.B., B.S., 1951 (Univ. Sydney), Wallsend District Hospital, Wallsend.

Latham, Geoffrey Rourke Welsford, M.B., B.S., 1951 (Univ. Sydney), Balmain and District Hospital, Balmain.

Marel, Joseph Oscar, registered in accordance with the provisions of Section 17 (1) (c) of the *Medical Practitioners Act*, 1938-1945, 143 Forbes Street, Darlinghurst.

Walker, John Bernard, M.B., B.S., 1951 (Univ. Sydney), 5 Day Avenue, Kensington.

The undermentioned have been elected as members of the New South Wales Branch of the British Medical Association:

Anderson, Gerard James, M.B., B.S., 1951 (Univ. Sydney), Lewisham Hospital, Lewisham.

Andrea, Brian Maxwell, M.B., B.S., 1951 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Bailey, Harry Richard, M.B., B.S., 1951 (Univ. Sydney), Flat 17, Capri Court, 1 Roslyn Street, Elizabeth Bay.

Blank, Joseph, registered in accordance with the provisions of Section 17 (1) (c) of the *Medical Practitioners Act*, 1938-1950, 4 Ormond Street, Bondi.

Blunt, John Littleton, M.B., B.S., 1951 (Univ. Sydney), Sydney Hospital, Sydney.

Brustolin, Nerino, M.B., B.S., 1951 (Univ. Sydney), Sydney Hospital, Sydney.

Bull, Alan Stuart, M.B., B.S., 1950 (Univ. Sydney), Prince Henry Hospital, Little Bay.

Bull, Richard Stuart, M.B., B.S., 1951 (Univ. Sydney), Broken Hill and District Hospital, Broken Hill.

Burfitt-Williams, Walter John, M.B., B.S., 1951 (Univ. Sydney), Lewisham Hospital, Lewisham.

Connors, John Joseph, M.B., B.S., 1948 (Univ. Sydney), 29 Thomas Street, Hurstville.

Cropley, David Oswald, M.B., B.S., 1951 (Univ. Sydney), Sydney Hospital, Sydney.

Cross, Douglas Oakeley, M.B., B.S., 1951 (Univ. Sydney), 43 Gordon Street, Balgowlah.

Dunn, John Talbot, M.B., B.S., 1951 (Univ. Sydney), Royal Newcastle Hospital, Newcastle.

Failes, David Geoffrey, M.B., B.S., 1951 (Univ. Sydney), Sydney Hospital, Sydney.

Ford, Bruce Francis, M.B., B.S., 1951 (Univ. Sydney), 12 Derby Street, Kogarah.

Gershon, Samuel, M.B., B.S., 1951 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Gibson, David Bruce, M.B., B.S., 1951 (Univ. Sydney), District Hospital, Camden.

Grant, Gordon, M.B., B.S., 1950 (Univ. Melbourne), Parramatta District Hospital, Parramatta.

Green, Ruth Helen, M.B., B.S., 1951 (Univ. Sydney), Sydney Hospital, Sydney.

Grosslicht, Robert, registered in accordance with the provisions of Section 17 (1) (c) of the *Medical Practitioners Act*, 1938-1950, 22 Cowper Street, Randwick.

Hallinan, Geoffrey Michael, M.B., B.S., 1951 (Univ. Sydney), 35 Webb Street, Croydon.

Harding, Bruce William Ian, M.B., B.S., 1951 (Univ. Sydney), Goulburn District Hospital, Goulburn.

Harrell, Francis Charles, M.B., B.S., 1951 (Univ. Sydney), Royal North Shore Hospital, St. Leonards.

Hodgkinson, Anthony Hugh Taylor, M.B., B.S., 1951 (Univ. Sydney), 16 Kitchener Street, Maroubra.

Jessup, Alan, M.B., B.S., 1951 (Univ. Sydney), Sydney Hospital, Sydney.

Johnson, Wilga Miriel, M.B., B.S., 1951 (Univ. Sydney), Lithgow District Hospital, Lithgow.

Jones, Bruce Littlewood, M.B., B.S., 1951 (Univ. Sydney), 41 Crandon Road, Epping.

Laszlo, John Eugene, registered in accordance with the provisions of Section 17 (1) (c) of the *Medical Practitioners Act*, 1938-1950, 17 Gladswood Gardens, Double Bay.

Lewis, Ronald Gordon, M.B., B.S., 1951 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Lyttle, Keith Wightman Purves, M.B., B.S., 1945 (Univ. Sydney), Derby Street, Walcha.

Mollenhauer, Paul Louis, registered in accordance with the provisions of Section 17 (2) of the *Medical Practitioners Act*, 1938-1945, 149 Castlereagh Street, Sydney.

Diary for the Month.

MARCH 6.—New South Wales Branch, B.M.A.: Executive and Finance Committee; Organization and Science Committee.
MARCH 6.—Queensland Branch, B.M.A.: Branch Meeting.
MARCH 7.—Victorian Branch, B.M.A.: Branch Meeting.
MARCH 7.—Western Australian Branch, B.M.A.: Council Meeting.
MARCH 8.—Queensland Branch, B.M.A.: Branch Meeting.
MARCH 9.—Queensland Branch, B.M.A.: Council Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney)—All contract practice appointments in New South Wales.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federal Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia; Medical Officer, South Australian Railways.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

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